INCREASE AND SPREAD OF BROWN SPOT NEEDLE BLIGHT WITHIN SINGLE AND MULTIPLE FAMILY PLANTINGS OF OPEN-POLLINATED LONGLEAF PINE

By

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"We shall find the real epidemic muddy and uncomfortable. And the explicit and logical analysis of our scattered observations is not only less uncomfortable than the statements of generalities, it also puts our cards on the table for all to see. Nevertheless, we must put up or shut up, striving constantly to use to the utmost the knowledge accumulated with such pain."

P. E. Waggoner

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By

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Longleaf pine (Pinus palustris Mill.) plantings were established in south Mississippi and southwest Georgia to study the increase in time and space of brown spot needle blight (BSNB), caused by Scirrhia acicola (Dearn.) Siggers. Open-pollinated families with varying degrees of resistance to BSNB were planted in single- and multiple-family plots. Epidemics were initiated by placing severely infected longleaf pine needles on susceptible longleaf source plants in the center of the plots. Disease was measured at 6-week intervals for two growing seasons. Disease progress curves were constructed from these measurements.

Lesion number progress curves fluctuated considerably during the course of the epidemics. Percent needle dieback progress curves proved more suitable to compare locations and families. Certain features of these curves (curve elements) were useful to describe the epidemic and

resistance among the families. Resistant families exhibited needle dieback 5-7 weeks later than the susceptible families and had lower maximum percent needle dieback levels. The rate of needle dieback/wk (YRATE) varied by distance from the initial infection focus but there were no significant family or location differences (YRATE = .026-.033).

Gradients of lesion numbers and percent needle dieback were steeper (more negative) in Georgia than in Mississippi. Both gradients flattened (became less negative) with time due to development of secondary foci. Ascospores of S. acicola probably contributed to the intensification of secondary foci as numerous ascospores were trapped at the beginning of the second growing season. The rate of pathogen movement outward from initial infection foci was similar for all families and locations. Isopaths of lesion numbers moved outwardly from center of plots at rates of 0.05 m/wk, while isopaths of needle dieback averaged 0.13 m/wk.

The mixing of resistant and susceptible families did not mitigate the BSNB epidemic as factors favoring within plant disease transmission predominated over those favoring between plant transmission. Family means of second-year height ranged 28-47 cm in Mississippi and 30-37 cm in Georgia. Correlation between maximum percent needle dieback and second year height at each location was not statistically significant (Mississippi: $r = 0.46$ and Georgia: $r = -0.45$).

INTRODUCTION

Brown spot needle blight (BSNB), caused by Scirrhia acicola (Dearn.) Siggers, has been a serious obstacle to the satisfactory regeneration and management of longleaf pine (Pinus palustris Mill.) for the past 50 years. The disease reduces plant vigor through defoliation, delays the onset of rapid height growth, and predisposes seedlings to early death. Such problems lead to extended rotation periods, irregular or inadequate stocking, and non-uniform stand conditions in plantations. As a result, longleaf is not planted extensively. Yet, in many areas, and particularly on sandy soils, longleaf grows as fast as other pines and has desirable traits that make it suitable for high value products. It is a preferred species for poles and lumber. Typical longleaf sites which are not successfully regenerated may be taken over naturally by loblolly (P. taeda L.) or slash (P. elliottii var elliottii Engelm.) pine or are planted to these species. Unfortunately, longleaf pine sites often have a large population of scrub oaks, and attempts to grow slash or loblolly pine shifts the major problem from BSNB to fusiform rust (caused by Cronartium quercuum (Berk.) Miyabe ex Shirai f. sp. fusiforme). Because of fusiform rust, there is renewed interest in longleaf pine.

The genetics of longleaf pine is a very important consideration for the successful control of BSNB. Longleaf possesses heritable resistance to BSNB, and research results have shown there is great opportunity for

improvement through genetic manipulation. It is important, therefore, to characterize and examine host-pathogen responses in both susceptible and resistant trees. Exactly how the resistance of different open-pollinated families of longleaf pine limit the development of BSNB in time and space is unknown. The comparison of disease increase and disease gradient curves for single family plantings and family mixtures may provide this information. Once families are characterized relative to the epidemic, it is possible to breed, propagate, and deploy them in a more intelligent manner.

The objectives of this study were to

1) Determine and compare the quantitative parameters of disease progress curves and disease gradient curves of artificially-induced BSNB epidemics in resistant, intermediate, and susceptible field plantings of open-pollinated longleaf pine.

2) Determine the effect that mixtures of resistant and susceptible families have on the quantitative parameters of disease progress curves and disease gradient curves of artificially-induced BSNB epidemics.

3) Compare the quantitative parameters of BSNB epidemics of resistant and susceptible families in two geographical areas--Mississippi and Georgia.

LITERATURE REVIEW

Host

Biology

Longleaf pine is one of the most distinctive species of the southern pines. At maturity it is a large tree, ranging from 24-37 m in height and averaging 61-76 cm in diameter at breast height. Individual trees typically possess a long, clear bole with a small open crown bearing dense tufts of needles, 25-45 cm in length, at the end of stout branches. The needles occur three to the fascicle, 3-sided, with each side bearing stomata. Typically, the root system consists of a deeply penetrating tap root with a wide-spreading and well-developed series of lateral roots. Longleaf pine is in the subsection Australes, which contains 11 species: eight in the southeastern United States, two in the West Indies, and one common to the West Indies and Central America (Little and Critchfield 1969).

Unlike other southern pines, longleaf pine seedlings possess a characteristic type of early development known as the "grass stage" (Wahlenberg 1946). This inherent short shoot pattern of growth is expressed upon seed germination and normally persists 2-5 years. If growing conditions are unfavorable the grass stage may last 20 years or more. Rapid stem elongation starts when the seedling attains a groundline diameter approaching 2.5 cm (Wahlenberg 1946) and a height of 6.5 cm (Siggers 1944). Conditions that inhibit seedlings from reaching

minimum diameter for active height growth are important. Once the tree is past the grass stage, however, longleaf grows rapidly and offers several desirable characteristics: excellent form, high specific gravity (Wahlgren and Schumann 1972), resistance to fire, and resistance to fusiform rust (Dinus 1974, Dorman 1976).

Newly-germinated longleaf seedlings have little or no above-ground hypocotyl, and the cotyledons are at groundline. The root tip, however, can extend 6 cm 7 days after germination (Allen 1965). Once beyond the grass stage, the stocky tap root may be over 1.5 m long, with a system of laterals 0.3-0.9 m below the surface and covering an area about 14 m^2 (Wahlenberg 1946).

Primary needles appear shortly after germination, followed by secondary needles two months later. Needle drop begins in July and continues through October. New needles develop continuously between March and September (usually 2-4 flushes occur, depending on rainfall amounts). As a result, the new needles of any one year vary in age and all do not mature and drop simultaneously. Needles persist about two years, the majority falling early in their third year (Heyward 1934).

Ecology

Longleaf pine occurs in a wide band extending along the Coastal Plain from Virginia to Texas, with extensions northward into the Appalachian foothills of Alabama and Georgia (Allen 1965). Sites vary from sea level to 610 m in northern Alabama.

Originally the longleaf pine belt was unbroken over extensive areas except for the moist bottomlands. This open, parklike virgin forest which occupied some 24 million ha is gone. Currently, there is less

than 5 million ha of second growth. Even this remnant is rapidly being destroyed because of ecological disturbances, aggravated by overcutting. These disturbances include changes in the frequency of burning, grazing of numerous hogs (Hopkins 1947a, b, c; Hopkins 1951; Peevy 1953) brown spot needle blight (Siggers 1944), and clearing of land for agriculture.

The longleaf pine type generally is not considered a climax in the strict sense (i.e., a community in essentially stable adjustment with soil and climate), but rather a fire subclimax (Chapman 1932, Wells and Shunk 1931): a successional stage that owes its long-time occupancy of extensive areas to frequent fires.

The species grows best on well-drained, acid, sandy soils, 0.9-24 m in depth (Allen 1965). On the flatlands along the coast, where soils are poorly drained, longleaf usually regenerates, but growth is very slow.

Longleaf pine is very intolerant of competition. Therefore, there are few stand associates. Pure, open stands are typical, with scattered hardwoods underneath, accompanied by a cover of numerous grasses. The common associates on dry sites are scrub oaks: blackjack (Quercus marilandica Muenchh.); bluejack (Q. incana Bartr.); turkey (Q. laevis Walt.); and saw palmetto (Serenoa repens (Bartr.) Small). On moist sites the common associates are slash pine, sweetgum (Liquidambar styraciflua L.), southern red oak (Q. falcata Michx. var. falcata), and loblolly pine. Species of Andropogon and Aristida (wire grasses) predominate.

Longleaf pine grows in warm, humid climates. It thrives where frequent, heavy summer rainfalls offset large losses of moisture caused by rapid percolation through and evaporation from sandy soils, and by transpiration during a long growing season (Woodward 1917). The annual rainfall throughout the longleaf belt ranges from 114-152 cm. The mean annual temperature range is 17-23 C (Wahlenberg 1946).

Management

The silvical requirements of and cultural practices applied to longleaf pine differ from those of other southern pines due to the nature of the seedling habit. Many consider the grass stage solely a deterrent to regeneration of the species by natural or artificial means. Many unsuccessful attempts to obtain adequate stocking and early height growth result from man's failure to recognize or appreciate the many ecological factors associated with the early growth and development of longleaf pine seedlings.

As early as 1883 it was recognized that longleaf stands most often failed to regenerate after logging and usually were succeeded by slash pine along the coast in the Southeast, and by loblolly pine or undesirable hardwoods further North and to the West (Hurt 1883, Vasey 1883). Fire is requisite to perpetuation of longleaf under natural conditions. Without frequent fires, hardwoods would dominate understories or other pines would outgrow the grass stage seedlings.

Until around 1935, wild grass and forest fires were generally regarded as an "unmitigated evil" in the South. Fortunately, the attitude of foresters towards woods burning in the longleaf range has changed dramatically (Wahlenberg 1946). Longleaf can withstand surface

fires except in the cotyledon and early grass stages. Two- or three-year-old seedlings possess a remarkable degree of heat resistance because of the morphological characteristics associated with their short shoot habit of growth; e.g., an exceptionally thick stem resulting in a well-protected cambium, in addition to numerous needle fascicles closely compacted upon the main axis which effectively enclose and protect the succulent apex. Seedlings become fire-resistant when the ground line diameter is approximately 0.8 cm (Bruce 1951, 1954). Immediately after height growth begins, the trees are again susceptible to fire (Hine 1925, Maple 1975, Siggers 1934). They usually gain height rapidly, however, and after 1.5 m or taller a light fire does no harm.

A major management problem of longleaf is irregular seed production. Good seed crops occur at 5- to 7-year intervals, failures one year out of five; but there are many exceptions to this periodicity (Wahlenberg 1946). Seed dispersal begins in October and is usually complete by the end of November. Unlike those of other southern pines, longleaf seeds germinate soon after dispersal in the fall. The seeds require contact with mineral soil for germination and establishment. Since the large seed (largest of the southern pines) and wing prevent them from penetrating heavy grass or litter, burning or mechanical measures to remove such barriers are essential (Chapman 1946, Croker 1957, Croker and Boyer 1975, Morriss and Mills 1948; Smith 1961).

Natural regeneration of longleaf pine requires 1) sufficient, desirable parent seed trees; 2) midstory and seedbed conditions favorable for establishment of seedlings; 3) protection from BSNB, hogs, and wildfire; and 4) complete release from overstory and brush

competition after adequate stocking has been secured (Croker and Boyer 1975). Three methods of natural regeneration--clearcutting, seed tree, and shelterwood--are successful for longleaf regeneration, particularly the latter (Croker and Boyer 1975).

When natural regeneration fails, artificial means are available. Direct seeding, perfected in 1957, succeeded on a wide array of sites (Derr and Mann 1959). Longleaf is the easiest of the southern pines to direct seed because the seed germinates quickly and mechanical seedbed preparation is often unnecessary (Mann 1969).

Greater success is achieved from planting 1-year-old longleaf nursery stock. Successful planting requires suitable-sized seedlings (>1.3 cm root collar diameter), high quality, careful handling, and proper length of storage (White 1979). The poor survival of 1-0 nursery seedlings is often due to a high density of seedlings grown per square meter in the nursery bed. Lower densities yield seedlings with thicker stems and a well-developed root system. The result is excellent survival.

The diversity of environments in the range of longleaf pine has fostered development and maintainence of a wealth of genetic variation among populations, or seed sources. Variations in soils, topography, and annual climate cause considerable variation among individual trees within populations. Mutation, migration, and introgression also contribute to variability within populations. Hence, longleaf pine is quite suitable for genetic manipulation (Snyder et al. 1977).

Gains in survival and growth are made through geographic selection (Bey and Snyder 1978, Wells and Wakeley 1970). Generally trees from the

Central Gulf Coast grow rapidly. Gains are achieved also by progeny testing in which individual parent trees are selected. For longleaf, phenotypic selection in wild populations is not the most effective system of improvement (Snyder 1969). The variable effects of BSNB infection, length of time in grass stage, competition, and fire mask the inherent growth of parents. When elite parents, the best 10 percent identified through progeny testing, are intercrossed, greater gains are made (Bey and Snyder 1978). Elite trees in natural stands can be crossed as well as elite trees in grafted seed orchards or the best trees in the best families in seedling seed orchards. Plantations from crosses among these elite trees also provide excellent opportunities for second generation selection. Thus, the potential for genetic improvement of longleaf pine is great.

Pathogen

Biology

History and taxonomy. The first collection of the fungus was on slash pine near Aiken, South Carolina, by H. W. Ravenel in 1876 (Hedgcock 1929, Little and Dorman 1954). De Thümen (1878) described Cryptosporium acicolum Thum. as the conidial stage for this specimen of Ravenel. It was placed in the imperfect family Melanconiaceae. Saccardo (1884) transferred it to the genus Septoria in the family Sphaeroidaceae on the basis of septate spores. The species appeared in Martin's (1887) list of the Septorias of North America as Septoria acericola (Thüm) Sacc. Undoubtedly, the specific name was misspelled due to a typographical error (Siggers 1944). In 1920 Saccardo (1920) described Actinothyrium marginatum on P. ponderosa Laws. from Idaho. Apparently this description was based on the fruit body of a species of

Leptostroma and the spores of another fungus, the latter being somewhat similar in gross structure to Saccardo's Septoria acicola (Thüm.) Sacc. (Sydow and Petrak 1924) and appearing in reddish lesions.

Sydow and Petrak (1922) described a new genus Lecanosticta, among the brown-spored Excipulaceace (Sphaeropsidales) with L. pini as the type species, from a collection on P. taeda in Arkansas. Later they studied the material of Ravenel and used the name Lecanosticta acicola (Thüm.) Syd and Petrak, and reduced L. pini to synonony with L. acicola. Sydow also obtained a specimen of A. marginatum and noted that the fungus in red areas and L. acicola were synonomous (Sydow and Petrak 1924).

Dearness (1928) emended the original description of the fungus. He found that the fungus was not a typical Cryptosporium, and that it fit better in this genus rather than Septoria. However, Hedgcock (1929) returned the species to Septoria, even though he realized it did not fit exactly. He also listed Petrak's (1922) L. decipiens as an additional synonym.

Siggers (1944) examined the A. marginatum collection Saccardo described, and stated the fungus in the red lesions was probably Dothistroma pini Hulbary. Finally, Siggers (1939, 1944) adopting Sydow's classification, preferred the genus Lecanosticta because of stromatic structure and colored spores, and designated L. acicola (Thüm.) Syd. as the imperfect stage.

Dearness (1926) first described the perfect stage of the pathogen as Oligostroma acicola from material collected near Silver Springs, Florida, by G. G. Hedgcock (Wolf and Barbour 1941). He placed it in the family Phyllachoraceae and the order Dothideales. He noted that the

perithecia were associated with the pycnidia of Cryptosporium acicolum and later (Dearness 1928) suggested that the two may be different stages of the same fungus. This was later proved by Siggers (1939) in laboratory experiments. Because of the perithecial stromata, however, he shifted the fungus from Oligostroma to Scirrhia, also at that time in the Phyllachoraceae. Wolf and Barbour (1941), working independently of Siggers, placed the perfect stage in Systremma Theiss. and Syd. and named the fungus Systremma acicola (Dearn.). Clements and Shear (1931) and Shear (1936), following the recommendations of the Cambridge revision of the International Code, retained Dothideae as the generic name and religated Systremma to synonomy.

Siggers (1944, p.11) placed the brown spot fungus in the genus Scirrhia, saying, "The ascigerous stage of the brown spot fungus . . . agrees well with Theissen and Sydow's [1915] emended description for Scirrhia Nits. . . . in stromatic characters . . .[the fungus] has more in common with Scirrhia rimosa (Alb. and Schw.) Fckl. [type specimen for Scirrhia] than with any of the other specimens studied." At that time Scirrhia was in the family Phyllachoraceae. Siggers (1944) expressed doubt the genus Scirrhia (non-clypeate) actually belonged in the Phyllachoraceae (clypeate). Indeed, Luttrell (1951), in his taxomonic revision of the Loculoascomycetes, placed Scirrhia from the Phyllachoraceae into the Dothidaceae. Wolf and Barbour (1941) and Siggers (1944) disagreed on the ascospore color: the former said there was color ("dilute brunnelois") and the latter described the spores as hyaline. Dearness (1926) also stated they were hyaline. Siggers (1944) showed they were hyaline, thereby permitting the fungus to be included in Scirrhia.

The list of synonyms is as follows:

Imperfect Stage:

Cryptosporium acicolum (Thüm.) Sacc.	1878
Septoria acicola (Thüm.) Sacc.	1884
Septoria acericola (Thüm.) Sacc.	1887
Actinothyrium marginatum (?) Sacc.	1920
Lecanosticta pini Syd. and Petrak	1922
Lecanosticta decipiens Petrak	1922
Lecanosticta acicola (Thüm.) Syd. and Petrak	1924

Perfect Stage:

Oligostroma acicola Dearness	1926
Scirrhia acicola (Dearn.) Siggers	1939
Systremma acicola (Dearn.) Wolf and Barbour	1941

The pathogen is currently classified as:

Class: Ascomycetes

Subclass: Loculoascomycetidae

Order: Dothidiales

Family: Dothideaceae

Genus: *Scirrhia*

Species: *Scirrhia acicola* (Dearn.) Siggers

Sporulation. Two types of spores are produced by S. acicola. The wind-blown ascospores cause long-distance spread, while the sticky, rain splash-disseminated conidia are reponsible for tree-to-tree spread and for disease increase on infected trees.

Two types of perithecium-like locules, spermogonia and carpogonia, are borne in a dark-colored ascostroma 2-3 months after the needle tissue has died (Wolf and Barbour 1941). Six to eight weeks later, mature pseudothecia form in the carpogonia. The asci adhere in a fascicle in each locule and do not mature simultaneously. Paraphyses are lacking. The asci grow upward as a group into sterile tissues which disintegrate as the asci develop. The ascus wall consists of two membranes. As a preliminary to ascospore discharge, the outer wall ruptures and the inner wall elongates in a "Jack-in-the-box" fashion. The ascospores are discharged one at a time in close succession through the ascus tip which has extended through the ostiolum to the outside. The ascospores are unequally bicellular, the upper cell being larger, and the spore wall is hyaline (Siggers 1944). Each cell contains two prominent brown oil globules. The spore measures 15-19 x 3.5-4.5μ Wolf and Barbour 1941). Ascospores are not found in the North Central States (Skilling and Nicholls 1974).

Conidial stromata (acervuli) form in necrotic mesophyll tissue within the needles. The stromata continue to increase in size as conidiophores form. The conidia are cylindical, curved, 1-3 septate, and brown. The pressure exerted by the mass of conidia produced by the acervuli lifts the overlying epidermis and hypodermis, rupturing them. Conidia are extruded from the fissures created by the rupture.

They are held together in a mucoid, water soluble matrix and accumulate in black heaps. Acervuli shed conidia indefinitely.

Hosts

Scirrhia acicola attacks 28 species of pine over a geographic range from southeastern United States into the North Central and Middle Atlantic States; in Oregon; and in Manitoba, Canada (Anonymous 1972, Laut et al. 1966, Nicholls and Hudler 1972, Nicholls and Skilling 1969, Phelps et al. 1978, and Siggers 1944). The pathogen is most destructive on longleaf pine seedlings in the southeastern United States, and on Scots pine (P. sylvestris L.) Christmas tree plantations in the North Central States (Nicholls and Skilling 1969, 1971; Nicholls et al. 1973; Prey and Morse 1971). The fungus also attacks slash and loblolly pine seedlings in nurseries (Siggers 1932, 1944).

The fungus varies in cultural characteristics, but these are not related to the percent infection on needles nor symptomology (Snow 1961). Isolates from longleaf were, in general, more infective than those from loblolly. Southern (conidial) isolates grew more rapid, had higher optimal temperatures for growth, and had higher germination percentages than those from northern locations (Kais 1972). In addition, southern isolates were more pathogenic on all species of pine examined: jack (P. banksiana Lamb.), sand (P. clausa (Chapm.) Vasey), Scots, loblolly, and longleaf pines.

Disease

C. W. Edgerton (Edgerton and Moreland 1924) at Louisiana State University in 1923, first demonstrated the pathogenicity of S. acicola using conidial suspensions of the fungus. E. C. Tims, also of Louisiana State University, obtained infection on slash and longleaf seedlings.

The fungus was reisolated from typical lesions (spots). However, neither Edgerton nor Tims published their results, (Siggers 1944). Therefore, the first published report showing the pathogenicity of this fungus was Hedgcock's work (1929).

This disease has several names. Chapman (1926) and several others called it "leaf rust." However, this implies a disease-causing basidiomycete in the Uredinales. Hedgcock (1929) used "brown spot disease" and stated others had called it "red spot." Siggers called the disease "brown spot needle blight" (1932, 1934) and "brown needle disease" (1939). Today the disease is known as "brown spot needle blight" or "brown spot."

Biology

Symptomology. The first symptom on longleaf pine is a small, grayish-green, circular spot which becomes yellow and later changes to light brown (Hedgcock 1929, Siggers 1944). A darker, chestnut brown discoloration often borders the spots (lesions) when acervuli form in the lesion (in cooler weather a purplish color borders the spots). Single infections will cause circular spots (3.2 mm in diameter) but contiguous infections coalesce to form irregular areas and a mottled appearance. Premature needle death is due to multiple spot not single spot infections. When the needle dies, the areas of green tissue between spots shrink more than the diseased areas. Then the needle has an embossed appearance (Siggers 1944).

Verrall (1934) described a second type of spot occurring more frequently on saplings than on grass stage plants. This "bar spot" is a a brownish spot on an amber-yellow band 3-5 mm long which is infiltrated with resin. As a result the lesion never matures fully. Verrall considered the bar spot symptom as evidence of resistance. Because bar spots are more prevalent on saplings (higher from ground) some believe these spots resulted from infection by the wind-disseminated ascopores (Siggers 1939, Verrall 1934).

Infected needles generally have three zones: a) the basal portion, which is usually green; b) the median portion, which is spotted with scattered lesions that alternate with green tissues; and c) the dead needle tips (Wolf and Barbour 1941). The upper end is usually the first infected because conditions favorable for the spread of the inoculum usually occur when growth is renewed in the spring and during the year.

As the tips of the needles are killed, they curve outward and down. By midwinter the dead needles, drooping around the stem, resemble a tussock of dead grass surmounted by the erect, standing spotted needles. As new needles are developed, each flush may be attacked severely. This results in stunting and, if significant defoliation occurs for three or more successive years, death of the plant occurs (Wolf and Barbour 1941).

Disease cycle on longleaf pine. Ascospores initiate the primary infection in the spring. They are produced on dead needles or dead portions of living needles. These needles occur on the plants or on the ground near the plants. Ascospores are most abundant in steadily increasing numbers from March to a peak in August (Kais 1971). They are forcibly ejected after rains, during fog, and dew periods, and are carried by the wind.

Conidia, produced in large numbers in the spring, may function as primary inoculum. They are released and disseminated during rains, but the largest numbers occur May through August (Kais 1971). The conidia are extruded in a water soluble, mucoid matrix, and adhere to the surface of the lesion until they are washed away by rain or dew.

Both spore types germinate via germ tubes. In culture, conidial germ tubes appear 14-52 hrs after being transferred to nutrient agar (Killebrew 1968, Siggers 1944, Wolf and Barbour 1941). Commonly, a germ tube grows from every cell of three-or four-celled conidia. However, on needles, the only germ tube grows from a terminal cell of the conidium (Patton and Spear 1978). Ascospores germinate at both ends, a germ tube being produced from each cell. The germ tubes grow appressed to the needle surface and follow the contours of the epidermis somewhat randomly (Killebrew 1968, Parris and Killebrew 1969).

The germ tube penetrates stomata (Patton and Spear 1978, Setliff and Patton 1974, Snow 1961, Wolf and Barbour 1941). A stimulus may attract a germ tube to a stoma, but this is not a general phenomenon expressed by all of the stomata (Patton and Spear 1978). After entering the antechamber of the stoma the germ tube grows irregularly without branching or becomes extensively branched and convoluted. No true appressorium or appressorium-like structure has been observed (Patton and Spear 1978). Penetration occurs between the guard cells and subsequent growth in the substomatal chamber or mesophyll. The hyphae within the leaf are initially intercellular. After a few days, however, the hyphae penetrate the thin walls of the mesophyll cells and the cells collapse

and die. The cell lumen quickly becomes filled with masses of hyphae. The vascular tissues are not invaded (Wolf and Barbour 1941). Symptoms are expressed 10 days to 11 weeks after penetration (incubation period), depending on environmental conditions (Boyce 1952, Hedgcock 1929, Kais 1977, Siggers 1944, Snow 1961). Within 10-14 days after the initial appearance of symptoms, the conidial stromata form and conidia are produced (latent period). Pseudothecial stromata form and ascospores are produced 2-3 months after the needle or portions thereof die (Wolf and Barbour 1941). Infections that occur during spring and summer are localized initially. During cool weather, however, the mycelium within lesions rapidly invades the living mesophyll tissue between the lesions and kills the needle (Siggers 1944). This results in the death of more tissue than is killed by the original lesion. Generally, newly emerging and succulent secondary needle tissue is more susceptible to infection (Kais 1977).

The fungus lives as a parasite for only a very short time. After the cell walls are penetrated and the cells killed, the fungus continues to live for many months in the dead needle tissue as a saprophyte (Epps 1959).

Numerous secondary cycles of infection begin in May and continue until the cool weather of late fall causes cessation of growth of the pathogen. The secondary inoculum is conidia. It is possible for ascospores to function as secondary inoculum if there is abundant needle kill early in the growing season. The cycle begins anew when the over-wintered pathogen in infected dead needles forms the primary inoculum source (ascospores) the following spring.

Ecology/Epidemiology

The primary environmental factor affecting the prevalence of BSNB on longleaf pine seedlings is moisture; i.e., rainfall, dew, or fog. The dissemination of conidia is dependent on splashing or wind-blown rain, while a film of free water on the needle surface is essential for spore germination (Siggers 1944, Verrall 1936). Extended moist periods are needed for conidial dissemination (Parris 1967, Siggers 1944, Verrall 1936). Kais (1971), however, found that the largest numbers of conidia were disseminated in heavy rainfalls and in rains which followed prolonged dry spells.

Temperature is the second most important environmental factor affecting BSNB. The optimun temperature for development of the fungus is 25 C, the maximum near 35 C, and the minimum between 5 and 10 C (Siggers 1944). Within this wide range temperature is not usually a limiting factor in disease development in the longleaf pine area, except in winter. Kais (1971) found conidia disseminated in rain at temperatures between 2-3 C. Ascospore discharge was greatest when the mean weekly temperatures exceeded 15 C, while numbers were low when temperatures were below 10 C. No ascospores were observed below 4 C. Conidia and ascospores are produced throughout the year (Henry 1954, Kais 1971), but the greatest numbers occur from April through August.

Kais (1975a) found that exposure to light following artificial inoculation enhanced infection, presumably by keeping the stomata open through which the fungus entered the needle tissue. Therefore, light may be a factor in greenhouse infection of longleaf. Kais (1977) also found that the susceptibility of fascicled needles decreased as needles elongated and matured.

Siggers (1932) observed that when competing vegetation was absent or sparse, disease severity was significantly worse. When ground cover was abundant, seedlings exhibited less disease. The ground cover probably reduced the amount of rain splashing and "caught" conidia. This reduced the number of conidia disseminated and the resultant infection.

Brown spot needle blight is a significant problem on longleaf pine throughout its range year-around, especially during mild winters, because 1) S. acicola spores are abundant and available year-round; 2) the fungus can grow and reproduce over a wide temperature range; 3) longleaf pine produces new needles flushes indeterminately; 4) longleaf pine has a juvenile grass stage close to the ground where humidity is high; and 5) longleaf pine requires bare mineral soil, i.e., no ground cover, for seed germination and seedling establishment.

BSNB is a chronic problem west of Panama City, Florida (Phelps and Kais 1975, Wakeley 1954), but this is probably not the only area. Kais (pers. comm.) states that infection is highly variable within short distances both within the heavy and light-to-moderate zones of infection (Phelps and Kais 1975). The distribution of BSNB depicted in their map is not founded on a current or systematic survey. Snyder and Derr (1972) reported low incidence in the eastern 60 percent of the range of longleaf pine although Kais (pers. comm.) disputes this report. Several other researchers in Federal, state, private, and university cooperatives have expressed opinions similar to Kais.

Management/Control

Fire. Wyman (1922) and Hedgcock (1929) first considered BSNB as a deterrent to regeneration of longleaf pine. Wyman credited H. W. Long, a pathologist with the Bureau of Plant Industry, with suggesting that fire might control BSNB. Ten years later, Chapman (1932) theorized that widespread fire suppression in the South, beginning around 1915, accounted for the increase of BSNB, which, prior to that time, was held in check by periodic fires. Although the use of fire was a controversial subject among foresters, studies by silviculturalists and plant pathologists confirmed the value of fire in BSNB control (Bruce 1951, Derr 1957, Siggers 1944, Wahlenberg et al. 1939, Wakeley and Muntz 1947, Wolf and Barbour 1941). Fire destroys infected needles, thus eliminating the source of inoculum for a few years. To avoid heavy mortality from burning, control should occur before BSNB incidence reaches 20-35 percent in midwinter (Croker 1967, Maple 1975). If percent infection is higher, losses from burning are increased because more dry, infected needles are attached to seedlings and on the ground. This creates higher temperatures of longer duration. In addition, burning is not recommended in the first growing season or when the trees are beginning height growth, as they are very suceptible to fire damage (Bruce 1951, Wahlenberg et al. 1939). Siggers (1934) suggested a single controlled winter fire after the second season of growth. Thereafter, controlled burning should be repeated at intervals of three years until height growth starts. Once height growth begins BSNB is not a serious problem since the trees grow out of the microclimate favorable for BSNB development. Fire is an effective tool when properly utilized. It is

an example of successful control of a high-infection-rate disease via sanitation (Schmidt 1978).

Chemical. Fungicides are an alternative for controlling BSNB. When Bordeaux mixture or lime-sulfur was applied at 14-day (Hedgcock 1929) or monthly (Wolf and Barbour 1941) intervals, excellent control was attained (Siggers 1932, 1933). However, except for nurseries, cost would be prohibitive. Later, Siggers (1944) reported good control in plantations with Bordeaux applications in the spring and fall for a two or three year period. Fungicidal control for areas of natural longleaf pine reproduction did not appear feasible.

Chlorothalonil (Bravo W-75) is effective and is registered for use against BSNB (Kais 1975b). Despite the effectiveness of this fungicide, chemical control for extensive forest areas is not economically feasible. Recently, Kais (1978) and Kais et al. (1981) found that the systemic fungicide, benomyl, administered as a root dip prior to planting, reduced the amount of BSNB for three years. Where inoculum was moderate, protection seemed adequate. A high concentration of benomyl (25% active ingredient) was phytotoxic. Judicious selection and application of systemic fungicides is a promising management tool. However, this has introduced a new problem: strains of plant-pathogenic fungi that are resistant to systemic fungicides are rapidly selected for in fungal populations exposed to those fungicides (Georgopoulos 1977). The cause is the metabolic specificity of the toxin action of the systemic fungicide. Fungicides which are potent and specific in their mode of action favor the

survival of resistant strains of the target fungus (Berger 1977; Dekker 1976, 1977; Georgopoulos 1977; Roberts 1978; Van der Hoeven and Bollen 1972). Public concern over a quality environment dictates that disease control be harmonious with the environment. Benomyl, unfortunately, is a mutagen and a teratogen in rats, and reduces earthworm populations in soil (Berger pers. comm.).

Genetics. A promising solution to controlling BSNB is the development of resistant longleaf pine strains through genetic manipulation. Genetic variation in resistance is extensive among seed sources. Seedlings from north Alabama remain disease-free when planted locally but are quite susceptible when grown near the Gulf Coast (Snyder and Allen 1968). In addition, seedlings from the central Gulf Coast are resistant at several locations (Bethune and Roth 1960, Derr 1971, Henry and Wells 1967, Snyder and Derr 1972) while seed sources from North Carolina and west of the central Gulf Coast generally are more susceptible and unpredictable (Bey and Snyder 1978).

Striking variation in resistance also occurs among individual trees. For example, disease-free seedlings were observed in infected areas (Derr 1963, Derr and Melder 1970), and, in an open-pollinated test involving 227 plus-trees from seven States, percentages of resistant seedlings per family ranged from 2 to 89 (Derr 1971). Crossing elite trees with other elite trees have yielded progeny with disease incidence one-half to one-third that of some open-pollinated, randomly selected trees (Bey and Snyder 1978). The genetic gain through selection and breeding is significant.

Comparisons among half-sib families in fungicide-sprayed and non-sprayed plots show that rapid early height growth is one, but not the only, mechanism of resistance (Snyder and Derr 1972). The correlation between the two plots was poor ($r = -0.22$). In addition, significant family x location interactions for brown spot resistance are reported (Bey and Snyder 1978). Selection for both resistance and growth is needed in an improvement program, and separate progeny tests are needed in each of the major areas where improved seed is to be used (Bey 1979).

An important aspect of genetic control of BSNB is genetic diversity. Without genetic diversity the results of selecting and breeding for disease resistance could be nullified (Schmidt 1978). Genetic homogeneity for disease resistance is quite hazardous for annual crops. Good examples are the southern corn leaf blight of 1970 and Victoria blight of 1946 on oats (Day 1974). Mass clonal production of a few resistant genotypes in forest trees likewise offers no buffer against the variability of the pathogen.

To combat genetic unformity, multiline cultivars were suggested by Jensen (1952) for oats and by Borlaug and Gibler (1953) and Borlaug (1959) for wheat to create the necessary intraspecific within-field diversity. This entailed mixing together several pure lines (or isolines), all identical except for a different gene or genes for specific (vertical) resistance. As a result, the increase of new races of the pathogen was delayed or prevented. Successess have been recorded for the oats/crown rust (Puccinia coronata Cda.) pathosystem in Iowa and contiguous states (Browning 1974, Browning and Frey 1969, Frey et al. 1973). Browning (1974) advocated using multilines which were aided by general (horizontal) resistance or tolerance.

Toxopeus (1956), Hooker (1967), Johnson et al. (1967), and Leonard (1969a, c) expressed concern about multiline varieties, as they might favor the development of complex races ("super races") of the pathogen. Jensen (1952), Allard and Bradshaw (1964), and Leonard (1969b) suggested that a multiline variety would be more effective if it consisted of a series of varieties with different genetic backgrounds rather than a series of backcross lines from the same recurrent parent variety in multilines. Wolfe and Barrett (1977), Wolfe (1978), and Wolfe et al. (1976), working with the barley/powdery mildew (Erysiphe graminis DC. f. sp. hordei Marchal) pathosystem, and Groenewegen and Zadoks (1979) using the wheat/yellow stripe rust (Puccinia striiformis West.) pathosystem preferred using mixtures of varieties rather than multilines. They said such mixtures provided a considerable degree of disease control even though the components were considered susceptible when grown in pure (or single) stands. In addition the mixtures were simpler and quicker to produce, and they added more heterogeneity to the environment. Several others (Browning et al. 1962, Berger 1973, Jensen and Kent 1963, Rothman and Frey 1953, and Suneson 1960) found that susceptible plants in mixed plantings with resistant plants were damaged less by disease than were susceptible plants in pure stands. In forestry there are no known examples of a tree having vertical resistance, matching a parasite with many races having specific pathogenicity. However, there is the opportunity to mix different families or clones. Variety mixtures approach more closely the situation in forest trees and the longleaf pine/S. acicola pathosystem.

Barrett (1978, 1980), Burdon (1978), Jeger et al. (1981, Kiyosawa (1977), Trenbath (1977), and Zadoks and Kampmeijer (1977) demonstrated the genetic consequences of using cultivar or variety mixtures through the use of theoretical ecological models and mathematical simulations of disease epidemics in such mixtures. They found that a mixture strategy slowed epidemic development and sustained high crop yields. Heybroek (1982, p.340), on the other hand, speaking in terms of forest stands, stated: "Broad generalizations on the effect of mixing of genotypes on the health of a stand or its components are dangerous. The effect may be different for each disease, site, host or case. Mixing may even be detrimental."

Functional diversity. Control or management of BSNB can be achieved also by observing the structure of natural forest ecosystems--they are functionally diverse. Such diversity plays a key role in determining the incidence and distribution of diseases in the forest (Schmidt 1978). Even-aged management in longleaf pine has fostered the increased incidence and spread of BSNB. The shelterwood silvicultural system as applied to longleaf pine (Boyer 1972, Boyer 1975, Croker and Boyer 1975, Maple 1977) is uneven-aged management. The overstory limits moisture on the understory trees, thus producing an unfavorable climate for the pathogen to multiply and spread. Since grass stage seedlings are fire-resistant, prescribed burning retards inoculum buildup from the outside during the time prior to overstory harvest (Boyer 1972). Following harvest of the overstory, the understory longleaf seedlings are sufficiently large to begin rapid

height growth in an environment unfavorable for disease development. Thus, capitalizing on the functional diversity provided by a forest stabilizes the incidence of BSNB without undue selection pressure on the pathogen.

Comparative epidemiology. In discussing the comparison of epidemics, Kranz (1974a) described several elements characteristic to disease progress curves; to list a few: D = the time at which the first disease symptoms are observed; X_o = the time from the beginning of host growth to the time the first discernable disease symptom is observed; Y_o = amount of disease in primary foci; and Y_{MAX} = maximum disease incidence. Curve elements are thought to represent certain biological events integrated in the disease progress curve. The elements are regarded as gauges for the effects of time and as a basis for quantitative comparisons of epidemics.

Kranz (1974a, b) has described several methods to compare entire disease progress curves in epidemics. Only two methods of comparison will be reviewed at they relate in this study. Van der Plank's (1963, 1965, 1967) analysis of the progress of epidemics in annual crops provides a tool to analyze the effects of single family and mixtures of resistant and susceptible families on disease increase. Van der Plank (1963) uses two methods to estimate r (rate of infection) from sample disease proportions. One method is by a linear regression on time (t) of the natural logarithm (ln) of the proportion of the plant population diseased (x) divided by the proportion not diseased ($1-x$). The slope of the regression line is taken as an estimate of r. The second method is similar, but uses disease proportions collected at only two times, t_1 and t_2. This is transformed again by $y = [x/(1-x)]$ and

$\underline{r}$ is estimated by $\underline{r} = (\underline{y}_2 - \underline{y}_1)/(\underline{t}_2 - \underline{t}_1)$. Statistical comparisons between $\underline{r}$-values using Van der Plank's first method has a major drawback: a normal distribution must be assumed. Unforunately, the distribution of $\underline{x}$ is unknown (Fulton 1979). Kranz (1974a, p.363) states: ". . . a serious shortcoming of these infection rates [$\underline{r}$-values] is that they do not provide for tests of significance." Waggoner (1965, p.120) said, "$\underline{r}$ is a function of environment and needs to change little to affect the course of disease profoundly." This consequence limits the suitability of $\underline{r}$ for comparison by classification. However, assuming that estimates of $\underline{x}$ follow a normal distribution, and a weighted regression analysis is performed (the variances under this assumption are nonhomogeneous; in linear regression, homogeneous variances are necessary for valid probability statements about the results), reliable estimates of $\underline{r}$ are obtained (Ashton 1972, Steel and Torrie 1960). The two-point method of Van der Plank gives reliable estimates of $\underline{r}$, but must be based on the means of several independent samples at each of the two points since the central limit theorem applies (Fulton 1979).

Another method (Griggs et al. 1978) to compare disease progress curves is a growth analysis procedure proposed by Rao (1965) and Wishart (1938). Disease progress curves are smoothed by fitting polynomials in time to the measurements for each tree (cumulative infection), the coefficients of the polynomial fit of each tree are substituted for the raw data, and the coefficients as response variables are compared via univariate and multivariate analyses of variance. This procedure allows statistical comparison of entire disease progress curves. Because of the method's flexibility it is applicable to all pathosystems.

To comparing disease gradient curves, Gregory (1968) proposed the following model:

$$\underline{y} = \frac{a}{\underline{X}^b}$$

where $\underline{y}$ is the expected quantity of disease or spores at distance $\underline{X}$ from a point source, b is the exponent of the gradient, and $\underline{a}$ is a constant. The model assumes that $\underline{y}$ is inversely proportional to some power of the distance $\underline{x}$. When $\log_{10} \underline{y}$ is plotted against $\log_{10} \underline{X}$ slopes (b) of different treatments can be compared. Disease gradients plotted with this function frequently flatten (slope decreases) over time (Gregory 1968, MacKenzie 1976, and Van der Plank 1963). Berger and Luke (1979) found that the $\log_{10} \underline{x}$ (= $\underline{y}$) transformation did not adequately straighten their disease gradient curves, and therefore, did not define the gradient when $\underline{x} > 0.5$. They successfully used the logit $\underline{x}$ [$f(\underline{x}) = \underline{x}/(1-\underline{x})$] transformation rather than $\log_{10} \underline{x}$. They found the flattening of disease gradients via the $\log_{10} \underline{x}$ transformation was more an aberration of the transformation than an actual change in the gradient rate.

MATERIALS AND METHODS

Longleaf Pine Selections

Open-pollinated seed was collected in 1977-1978 from ramets (grafts of original parent tree) of several longleaf pine selections belonging to Region 8 (R-8) of the National Forest System, U. S. Forest Service, and one Southern Forest Experiment Station (SFES) selection. Ramets of all R-8 selections are located in R-8 seed orchards in Mississippi, South Carolina, and Louisiana (Table 1). The SFES selection is a graft located on the J. K. Johnson Tract of the U. S. Forest Service's Palustris Experimental Forest in Alexandria, Louisiana.

Open-pollinated progeny of parents MS-6 and MS-14 are resistant (R) to BSNB, MS-4 and SC-6 are intermediate (I), and MS-5, SC-26, TX-1, TX-23, and Stu-4 are susceptible (S) to BSNB (Table 1) (Snyder and Hamaker 1978, Snyder 1977, Derr 1971). Classification of these families was based on visual estimates of the needle area killed by BSNB as a percentage of the total needle area on a plant.

Growing Seed

Seed was sown in nursery beds at the U. S. Forest Service's W. W. Ashe Nursery in Brooklyn, Mississippi, in October, 1978. Spacing was 15 cm between drills and 5 cm within drills, or 226 plants per m^2 (21 plants per ft^2). Seedlings received normal nursery culture: weeding and watering. The fungicide chlorothalonil was sprayed on

Table 1. Longleaf pine selections used to determine the increase and spread of Scirrhia acicola within single and multiple family treatments of longleaf pine.

Selection	Brown spot needle blight rating	Location of ramets
MS-4	Intermediate	Erambert Seed Orchard, Hattiesburg, MS.
MS-5	Susceptible	Erambert Seed Orchard, Hattiesburg, MS.
MS-6	Resistant	Erambert Seed Orchard, Hattiesburg, MS.
MS-14	Resistant	Erambert Seed Orchard, Hattiesburg, MS.
SC-6	Intermediate	Francis Marion Seed Orchard, Monck's Corner, SC.
SC-26	Susceptible	Francis Marion Seed Orchard, Monck's Corner, SC.
TX-1	Susceptible	Stuart Seed Orchard, Pollock, LA.
TX-23	Susceptible	Stuart Seed Orchard, Pollock, LA.
Stu-4	Susceptible	Palustris Experimental Forest, Alexandria, LA.

seedlings to protect them from BSNB infection. They were fertilized three times during the growing season and top-pruned twice. Eight weeks prior to lifting all seedlings were undercut (root-pruned). Seedlings were lifted and transplanted to field plots in January, 1980. Replacement seedlings were kept in 0.9 l milk cartons.

Plantation Establishment

Sites

Identical plantings were established at two locations: in south Mississippi on the U. S. Forest Service Harrison Experimental Forest (HEF), in Harrison County, near Saucier; and in southwestern Georgia on International Paper Company's Southlands Experimental Forest (SEF), Decatur County, just south of Bainbridge. Both planting locations were "typical longleaf pine sites": well-drained with deep sands. The sites were burned in the winter and double-disked prior to plantation establishment. Volunteer longleaf pine seedlings in and around the planting sites were removed and destroyed before planting.

Experimental Design

The experimental design was a randomized complete block design with eleven treatments, four replications, and two locations. The eleven treatments were: six single families, four multiple families, and one control (Table 2). Each multiple family treatment consisted of alternately-planted seedlings from a resistant and a susceptible family. Four combinations were possible (Table 2). The control was an equal seed mix of three susceptible families: TX-1, TX-23, and Stu-4 (Table 1,2). Each treatment was a square plot of 64 seedlings (Fig. 1, 2) in

Table 2. Single and multiple family treatments of open-pollinated longleaf pine used to determine Scirrhia acicola increase and spread over time.

Treatment [family(s)]	BSNB Rating[a]
MS-6	R
MS-4	I
MS-5	S
Non-inoculated control [b]	S
MS-14	R
SC-6	I
SC-26	S
MS-6 + MS-5	R + S
MS-14 + MS-5	R + S
MS-6 + SC-26	R + S
MS-14 + SC-26	R + S

[a] R, I, and S = resistant, intermediate, and susceptible to brown spot needle blight (BSNB), respectively.

[b] A mixture of open-pollinated seed from three susceptible longleaf pine parents: TX-1, TX-23, and Stu-4.

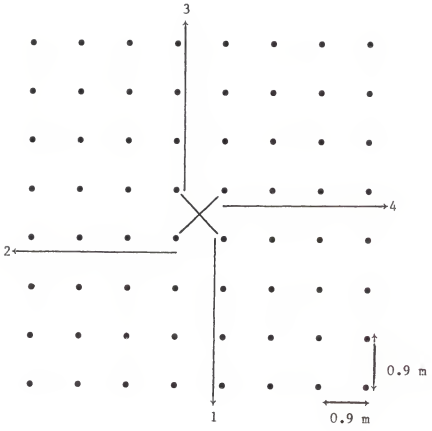


Fig. 1. Single family treatment plot of 64 longleaf pine seedlings to determine spread of Scirrhia acicola in Mississippi and Georgia. Epidemic was initiated in center of each plot on spreader trees (X). Arrows from center of plot depict seedlings along the four transects that were measured.

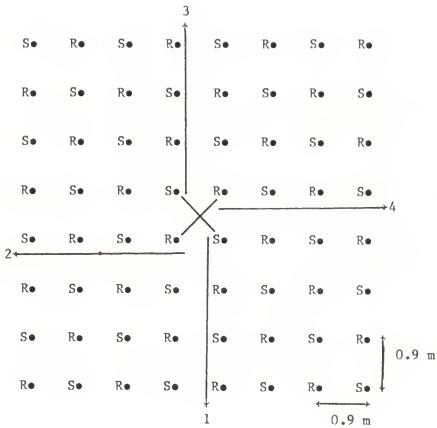


Fig. 2. Multiple family treatment plot of 64 longleaf pine seedlings to determine spread of Scirrhia acicola in Mississippi and Georgia. Epidemic was initiated in center of each plot on spreader trees (X). Arrows from center of plot depict seedlings along the four transects that were measured. (S = individual of a susceptible family; R = individual of a resistant family.)

in each of the four replications at both locations. Spacing within each plot was 0.9 x 0.9 m. Surrounding each square plot was a 6-m-wide grass strip to reduce interplot interference of inoculum due to rain splash.

Inoculum

During plantation establishment eight seedlings from the susceptible seedlot mix were planted in the center of each treatment plot with the exception of the control treatment plots. These seedlings were "spreaders" for the disease and were spaced 0.3 m apart (Fig. 1, 2). Epidemics were initiated in each plot by placing severely infected needles on the "spreader" plants in May 1980 after newly-expanding needles were approximently 6 cm long. The severely infected needles (inoculum) were collected from a natural stand (1 ha) of heavily infected 2-to 3-yr-old longleaf pine seedlings and saplings on the HEF. Collection of needles was random throughout the 1 ha stand.

Plantation Maintenance

Weeds were removed by hoeing within each square plot to allow maximum spread of *S. acicola* inoculum by rain splash. Weeds were not removed from the 6 m wide buffer zone surrounding each square plot. The buffer zones were sown with millet seed.

Weather

Relative humidity and air temperature were continuously monitored at both locations with a recording hygrothermograph placed in a 1.2 m-tall weather shelter in the center of the plantation. Precipitation was measured with a recording rain gauge.

Spore Trapping

At the Mississippi field location a Kramer-Collins 24-hour continuous sampling spore trap was positioned 0.3 m above the ground in the center of the planting to determine if ascospores of S. acicola were being wind-disseminated at the site. The spore trap sampled 28 l of air per minute four times every hour. Spores were collected on a cellulose liquid adhesive-greased microscope slide. Slides were replaced every 24 hr and counted Monday-Saturday for the duration of the study.

To catch conidia, vaseline-coated glass slides were placed at distances of 0, 0.1, 0.3, 0.6, 0.9, and 1.2 m from four randomly selected trees. These slides were replaced and conidia counted every week for 4 weeks during the late summer of 1981.

Pathometry

When symptoms were first observed 5 to 6 weeks after plot inoculation on seedlings next to the spreaders, disease measurements began at both plantings and continued for the next year-and-a-half at 6-week intervals. The control plots were assessed to measure background inoculum at each location.

Measurements were taken along four transects (one transect in each compass direction in each plot): S, W, N, and E for the Mississippi planting and SW, NW, NE, and SE for the Georgia planting), with four trees per transect (Fig. 1, 2). Sixteen seedlings were measured in each treatment plot. The four trees within a transect were 0.3, 1.2, 2.1, and 3.0 m from the plot center. Ten fascicles (1 fascicle = 3 needles) on the side closest to the disease spreaders and ten fascicles on the side opposite (180°) the disease spreaders were measured on each sample tree. Measurements taken on each side of a sample tree were the

fascicle length (cm), number of fascicles infected with BSNB, number of fascicles dead from BSNB, number of fascicles healthy, total number of BSNB lesions on the needles of five fascicles, and the needle area dead from BSNB as a percentage of the area of the ten fascicles measured (=percent needle dieback). Measurements on both sides were used to obtain values for the sample tree. Fascicle length and percent needle dieback (each) were averaged, while the number of fascicles infected, dead, and healthy and the number of BSNB lesions were each summed to give values for the sample tree. Measurements taken on the whole sample tree were survival and height to the nearest 0.5 cm from groundline to tip of bud.

Analyses

Analysis of variance of means for all measurements of individual trees and plots for the randomized complete block design by distance within location (split plot), by location (split split plot), and over both locations was used to test differences among treatments. Treatment plots were the main plot, transects the sub plot, and distance from the center of the inoculated plot the sub sub plot (Table 3). When applicable, tests of significance (Duncan's multiple range test) were performed at the 0.05 level. Analyses were performed with single family treatments followed by inclusion of the multiple family treatments. The non-inoculated control was not included in the analyses.

Disease Increase

Lesion number. The rate of increase of lesion numbers was followed by plotting total number of lesions vs time after epidemic initiation. Single and multiple family curves were drawn for distance from initial infection focus.

Table 3. Analysis of variance tables and expected mean squares for brown spot variables in a randomized complete block design using single and multiple family treatments of longleaf pine.

By distance from inoculum within location (split plot):

Source	d.f.	Expectation of mean squares
Blocks ¶	(b-1)	$\sigma^2_{e_2} + jk\sigma^2_b$
Treatments	(b-1)	$\sigma^2_{e_2} + ik\tau^2_f$
Error 1	(b-1)(f-1)	$\sigma^2_{e_2}{}^r$
Transects	(t-1)	$\sigma^2_{e_1} + ij\tau^2_t$
Treatment x transect	(f-1)(t-1)	$\sigma^2_{e_1} + i\tau^2_{ft}$
Error 2	f(b-1)(t-1)	$\sigma^2_{e_1}{}^s$

By location (split split plot):

Source	d.f.	Expectation of mean squares
Blocks	(b-1)	$\sigma^2_{e_3} + jkm\sigma^2_b$
Treatments	(f-1)	$\sigma^2_{e_3} + ikm\tau^2_f$
Error 1	(b-1)(f-1)	$\sigma^2_{e_3}{}^t$
Transects	(t-1)	$\sigma^2_{e_2} + ijm\tau^2_t$
Treatment x transect	(f-1)(t-1)	$\sigma^2_{e_2} + im\tau^2_{ft}$
Error 2	f(b-1)(t-1)	$\sigma^2_{e_2}{}^u$

Table 3--continued.

By location-continued:

Source	d.f.	Expectation of mean squares
Distances	(d-1)	$\sigma^2_{e_1} + ijk\tau^2_d$
Treatment x distance	(f-1)(d-1)	$\sigma^2_{e_1} + ik\tau^2_{fd}$
Transect x distance	(t-1)(d-1)	$\sigma^2_{e_1} + ij\tau^2_{td}$
Treatment x transect x distance	(f-1)(t-1)(d-1)	$\sigma^2_{e_1} + i\tau^2_{ftd}$
Error 3	(b-1)(d-1)+ (b-1)(t-1)(d-1)+ (b-1)(f-1)(d-1)+ (b-1)(f-1)(t-1)(d-1)	$\sigma^2_{e_1}{}^v$

Over both locations:

Source	d.f.	Expectation of mean squares
Locations	(l-1)	$\sigma^2_{e_4} + ijkm\tau^2_l$
Blocks (locations)	l(b-1)	$\sigma^2_{e_4}{}^w$
Treatments	(f-1)	$\sigma^2_{e_3} + pikm\tau^2_f$
Location x treatment	(l-1)(t-1)	$\sigma^2_{e_3} + ikm\tau^2_{lf}$
Error 2	l(b-1)(f-1)	$\sigma^2_{e_3}{}^x$
Transects	(t-1)	$\sigma^2_{e_2} + pijm\tau^2_t$
Location x transect	(l-1)(t-1)	$\sigma^2_{e_2} + ijm\tau^2_{lt}$

Table 3--continued.

Over both locations-continued:

Source	d.f.	Expectation of mean squares
Treatment x transect	(f-1)(t-1)	$\sigma^2_{e_2} + pim\tau^2_{ft}$
Location x family x transect	(l-1)(f-1)(t-1)	$\sigma^2_{e_2} + im\tau^2_{lft}$
Error 3	l(b-1)(t-1)+l(b-1)(f-1)(t-1)	$\sigma^2_{e_2}$ y
Distances	(d-1)	$\sigma^2_{e_1} + pijk\tau^2_{d}$
Locations x distance	(l-1)(d-1)	$\sigma^2_{e_1} + ijk\tau^2_{ld}$
Treatment x distance	(f-1)(d-1)	$\sigma^2_{e_1} + pik\tau^2_{fd}$
Transect x distance	(t-1) (d-1)	$\sigma^2_{e_1} + pij\tau^2_{td}$
Location x treatment x distance	(l-1)(f-1)(d-1)	$\sigma^2_{e_1} + ik\tau^2_{lfd}$
Location x transect x distance	(l-1)(t-1)(d-1)	$\sigma^2_{e_1} + ij\tau^2_{ltd}$
Treatment x transect x distance	(f-1)(t-1)(d-1)	$\sigma^2_{e_1} + pi\tau^2_{ftd}$
Location x treatment x transect x distance	(l-1)(f-1)(t-1)(d-1)	$\sigma^2_{e_1} + i\tau^2_{lftd}$

Table 3--continued.

Over both locations-continued:

Source	d.f.	Expectation of mean squares
Error 4	1(b-1)(d-1)+ 1(b-1)(f-1)(d-1) +1(b-1)(t-1)(d-1) +1(b-1)(f-1)(t-1) (d-1)	$\sigma^2_{e_1}$ [z]

[q] All effects are fixed except the effect of blocks; p, i, j, k, and m = number of locations, blocks, treatments, transects, and distances, respectively.

[r] $\sigma^2_{e_2} = \sigma^2_e + k m \sigma^2_{bf}$

[s] $\sigma^2_{e_1} = (\sigma^2_e + j\sigma^2_{bt}) + \sigma^2_e$

[t] $\sigma^2_{e_3} = \sigma^2_e + k m \sigma^2_{bf}$

[u] $\sigma^2_{e_2} = (\sigma^2_e + jm\sigma^2_{bt}) + (\sigma^2_e + m\sigma^2_{bft})$

[v] $\sigma^2_{e_1} = (\sigma^2_e + jk\sigma^2_{bd}) + (\sigma^2_e + k\sigma^2_{bfd}) + (\sigma^2_e + j\sigma^2_{btd}) + \sigma^2_e$

[w] $\sigma^2_{e_4} = (\sigma^2_e + pjkm\sigma^2_b) + (\sigma^2_e + jkm\sigma^2_{lb})$

[x] $\sigma^2_{e_3} = (\sigma^2_e + pkm\sigma^2_{bf}) + (\sigma^2_e + km\sigma^2_{lbf})$

[y] $\sigma^2_{e_2} = (\sigma^2_e + pjm\sigma^2_{bt}) + (\sigma^2_e + jm\sigma^2_{lbt}) + (\sigma^2_e + pm\sigma^2_{bft}) + (\sigma^2_e + m\sigma^2_{lbft})$

[z] $\sigma^2_{e_1} = (\sigma^2_e + pjk\sigma^2_{bd}) + (\sigma^2_e + jk\sigma^2_{lbd}) + (\sigma^2_e + pi\sigma^2_{bfd}) + (\sigma^2_e + k\sigma^2_{lbfd}) +$
$(\sigma^2_e + pk\sigma^2_{btd}) + (\sigma^2_e + j\sigma^2_{lbtd}) + (\sigma^2_e + p\sigma^2_{bftd}) + \sigma^2_e$

Percent dieback. *Polynominal curve fitting.* Disease progress curves of percent dieback (y_t) for each of the 704 trees (per measurement period and location) were fitted with smooth polynominals in time (t). The form of the polynominal for each tree was

$$y(t) = b_o + b_1 t + b_2 t^2 + b_3 t^3 + \ldots b_p t^p$$

where y(t) was the predicted dieback at time t and the b's were the polynominal coefficients estimated by least squares. The deviations from prediction (errors) were assumed to be distributed normally with mean zero and common variance. The degree of each polynominal (p) was the same for each tree. The term b_o represents the amount of dieback on a tree at the time the plots were first inoculated--zero because only brown spot-free seedlings were used. Therefore, b_o was set to zero and the fitting procedure adjusted accordingly. An analysis of variance for randomized complete block design by distance within location (split split plot) and by location (split plot) was performed on the b_p's ($P = 0.05$).

Curve elements. Initially polynominals were thought to be adequate to describe the disease (percent dieback) progress curves for BSNB,, however, they were not sufficient. Therefore, another approach was taken: various disease progress curve elements were compared by methods presented by Kranz (1974a). The following elements were recorded for each tree over the two growing seasons:

YMAX - the maximum amount of percent BSNB dieback observed.

TBEG - the time (wk) when percent BSNB dieback was first observed.

TMAX - the time (wk) when YMAX was observed.

YRATE - the nontransformed rate of percent BSNB dieback increase per week:

$$YRATE = \frac{YMAX - YMIN^*}{TMAX - TBEG}$$

*YMIN = 0

An analysis of variance of the data for each curve element was performed by distance within location (split split plot), by location (split plot) and over locations (Table 3). Significance tests were performed at the 0.05 level.

Disease Gradients

Gradients for both lesion numbers and percent dieback were obtained by plotting logit ($\underline{x}$ + 0.05) or $\log_{10}$ ($\underline{x}$ + 0.05) [$\underline{x}$ = proportion dieback] vs $\log_{10}$ d (d = meters). Slopes of disease gradients were subjected to analyses of variance (Table 4). Tests of significance were performed at the 0.05 level.

Rate of Spatial Disease Spread

The rate of BSNB spread in space was calculated from plotted disease progress curves for lesion numbers and percent dieback observed at 0.3 and 3.0 m from the plot centers for various periods. The interlinear distance (2.7 m) was divided by the number of weeks needed for lesion numbers or percent dieback at 0.3 m to reach the same severity at 3.0 m.

Table 4. Analysis of variance table and expected mean squares used for disease gradient slopes of Scirrhia acicola on single and multiple family treatments of longleaf pine.

Source	d.f.	Expectation of mean squares
Blocks	(b-1)	$\sigma_e^2 + j\sigma_b^2$
Treatments	(f-1)	$\sigma_e^2 + i\tau_f^2$
Error	(b-1)(f-1)	σ_e^2

RESULTS

Planting Survival

Planting survival was excellent at both locations. The Mississippi planting had a mean survival of 98.3% with a range of 94.7-100%. The Georgia planting had a mean survival of 96.3% with a range of 92.6-99.6%. The few seedlings that died prior to the inoculation of the plots were replaced successfully with healthy, vigorous, containerized seedlings so that when the epidemic was initiated all plots had 100% tree survival.

Tree Height

By the end of the second year 100% of the trees at the Mississippi planting were out of the grass stage (>7.5 cm), while at the Georgia planting 96% of the trees were out of the grass stage.

Height was significantly affected by distance from the initial infection focus at both locations. Therefore, data were analyzed by distance.

Mississippi

Family effects were significant only at the closest distance (0.3 m) (Table 5). The resistant family MS-14 had the shortest trees while the susceptible family, MS-5, had the tallest. However, the other susceptible family, SC-26, had the second shortest trees while the other resistant family, MS-6, had the second tallest (Table 6). Family means over all distances ranged 28.9 cm to 46.6 cm. The control was the

Table 5. Analysis of variance tables with mean squares for disease variables and height at four distances for six single family treatments of longleaf pine exposed to Scirrhia acicola for two growing seasons in Mississippi.

Source	d.f.	YMAX [a] Distance (m)				TBEG [c] Distance (m)				TMAX [d] Distance (m)			
		0.3	1.2	2.1	3.0	0.3	1.2	2.1	3.0	0.3	1.2	2.1	3.0
Blocks	3	0.3	16.3	17.2	30.8	8.3	3.8	6.5	4.5	3.4	2.6*	1.5	1.3
Treatments	5	1.1	28.3*[b]	63.3*	43.0*	3.8	3.7	4.5	19.7*	1.3	0.6	0.8	0.7
Error 1	15	0.5	7.3	8.0	11.4	4.2	6.2	5.3	2.9	1.8	0.6	1.1	0.6
Transects	3	0.4	16.8*	11.3	19.6	8.2*	11.6	16.3*	12.8*	4.2	1.2	0.7	0.4
Treatment x transect	15	0.3	8.8*	11.6*	9.9	1.7	6.7	8.7	12.6*	1.8	1.0	0.4	0.6
Error 2	54	0.6	3.9	5.2	7.3	1.9	4.8	5.2	4.6	1.2	0.9	0.5	0.8

Source	d.f.	YRATE [e] Distance (m)				LESION NO. [f] Distance (m)				HEIGHT [g] Distance (m)			
		0.3	1.2	2.1	3.0	0.3	1.2	2.1	3.0	0.3	1.2	2.1	3.0
Blocks	3	.02	2.3	4.5	2.9	3699	103.5	3.4	7.6	248.2	861	1399	333.7
Treatments	5	.27	1.0	4.5	2.2	1724	176.3	21.9	5.0	918.2*	751	1559	566.4
Error 1	15	.21	2.5	2.9	1.5	2810	80.2	23.0	17.7	303.9	1051	700	418.5
Transects	3	.29	2.9	5.7	2.1	3734*	124.2	19.8	34.4	45.3	264	379	125.3
Treatment x transect	15	.19	1.2	3.9	5.0	600	65.6	19.2	16.0	320.2*	533	330	75.8
Error 2	54	.14	1.7	2.6	1.6	949	64.8	22.4	19.0	138.7	293	324	240.3

Table 5--extended.

[a] Maximum percent needle dieback.

[b] Asterisks indicate the probability (P=0.05) of a value larger than F.

[c] Time (wk) when needle dieback was first observed.

[d] Time (wk) when YMAX was observed.

[e] Rate of percent needle dieback increase per week.

[f] Lesion numbers observed 18 wk after epidemic initiation.

[g] Second-year height (cm).

Table 6. Means by distance from initial infection focus of disease variables and height for single and multiple family treatments of longleaf pine exposed to Scirrhia acicola for two growing seasons in Mississippi.

Treatment	Distance (m)	YMAX[ry] (%)	TBEG[sy] (wk)	TMAX[t] (wk)	YRATE[u]	LESION[v] NO.	HEIGHT[wy] (cm)	b_1[x]
MS-6 (R)[q]	0.3	99	31.8	70.8	.027	30.2	38.9ab	.235
MS-4 (I)		93	25.8	72.0	.022	58.4	34.9abc	.303
MS-5 (S)		100	29.4	67.8	.027	51.9	42.3a	.525
MS-14 (R)		97	31.8	70.8	.026	40.6	23.9c	.254
SC-6 (I)		100	25.8	68.4	.025	38.6	27.8bc	.361
SC-26 (S)		99	26.4	68.4	.025	50.5	25.6c	.391
MS-6 + MS-5		99	32.4	71.4	.027	29.2	44.4	.361
MS-14 + MS-5		96	28.8	69.6	.025	42.2	39.2	.184
MS-6 + SC-26		95	28.8	69.6	.026	41.0	36.3	.220
MS-14 + SC-26		98	23.4	67.2	.024	57.9	24.9	.392
Control		63	66.6	77.4	.053	1.3	43.4	0
$\bar{x}$[z]		98	28.2	69.6	.025	44.1	33.8	.323
MS-6 (R)[q]	1.2	81ab	47.4	72.6	.041	4.6	44.9	.042
MS-4 (I)		70bc	45.6	72.0	.036	9.3	46.2	.055
MS-5 (S)		88ab	44.4	73.2	.035	10.9	44.8	.087
MS-14 (R)		57b	45.0	75.6	.029	3.3	29.8	.017
SC-6 (I)		80ab	44.4	72.6	.031	3.8	34.1	.083
SC-26 (S)		94a	39.0	72.6	.032	9.2	36.7	.134
MS-6 + MS-5		76	45.6	75.6	.025	5.5	37.8	.045
MS-14 + MS-5		71	42.6	74.4	.026	7.8	39.5	.024
MS-6 + SC-26		79	47.4	76.2	.035	6.3	35.8	.010
MS-14 + SC-26		87	40.8	72.6	.036	16.6	28.4	.140

Table 6--continued.

Treatment	Distance (m)	YMAX[ry] (%)	TBEG[sy] (wk)	TMAX[t] (wk)	YRATE[u]	LESION[v] NO.	HEIGHT[wy] (cm)	b_1[x]
Control	1.2	59	60.6	74.4	.052	1.8	48.3	.003
$\bar{x}$[z]		78	44.4	73.8	.032	7.7	37.8	.064
MS-6 (R)[q]	2.1	59bc	58.8	75.6	.044	2.1	51.2	.004
MS-4 (I)		40cd	55.2	73.2	.028	1.8	40.3	.010
MS-5 (S)		77ab	52.2	75.6	.039	3.1	52.8	.022
MS-14 (R)		36d	52.8	76.8	.026	2.4	28.1	.032
SC-6 (I)		59bc	54.6	75.0	.048	4.9	37.6	.019
SC-26 (S)		87a	49.2	77.4	.042	3.8	32.8	.032
MS-6 + MS-5		59	59.4	74.4	.046	2.9	48.6	.021
MS-14 + MS-5		48	59.4	77.4	.032	2.8	37.9	.009
MS-6 + SC-26		73	55.2	76.2	.041	4.8	30.3	.002
MS-14 + SC-26		76	47.4	75.6	.039	4.4	36.8	.038
Control		56	57.6	76.8	.036	1.5	49.4	.014
$\bar{x}$[z]		61	54.6	75.6	.038	3.3	39.6	.019
MS-6 (R)[q]	3.0	45ab	52.8b	75.6	.020	3.2	44.9	.049
MS-4 (I)		42ab	52.2b	75.6	.035	3.4	45.1	.010
MS-5 (S)		60a	51.6b	76.2	.031	3.4	46.3	.048
MS-14 (R)		27b	62.4a	77.4	.023	2.1	33.6	.003
SC-6 (I)		29b	65.4a	74.4	.028	3.4	37.5	0
SC-26 (S)		68a	49.2b	73.8	.034	2.6	33.7	.030
MS-6 + MS-5		43	53.4	76.8	.031	2.8	42.5	.036
MS-14 + MS-5		51	55.2	76.2	.042	3.4	40.1	.055
MS-6 + SC-26		64	55.8	77.4	.035	2.1	35.0	.010

Table 6--continued.

Treatment	Distance (m)	YMAX[ry] (%)	TBEG[sy] (wk)	TMAX[t] (wk)	YRATE[u]	LESION NO.[v]	HEIGHT[wy] (cm)	b_1[x]
MS-14 + SC-26	3.0	66	48.6	75.0	.031	2.2	29.5	.080
Control		51	61.2	76.2	.040	3.2	46.8	.022
$\bar{x}$[z]		50	54.6	75.6	.031	2.9	38.8	.032

[q] R, I, and S = resistant, intermediate, and susceptible to brown spot needle blight, respectively.

[r] Maximum percent needle dieback.

[s] Time (wk) when needle dieback was first observed.

[t] Time (wk) when YMAX was observed.

[u] Rate of percent needle dieback increase per week.

[v] Lesion numbers observed 18 wk after epidemic initiation.

[w] Second-year height (cm).

[x] Linear coefficient of a fitted polynomial based on the first growing season percent needle dieback.

[y] Numbers followed by the same letter are not significant (P=0.05) according to Duncan's multiple range test. Multiple family treatments were not included in this test.

[z] Mean of single family treatments only.

tallest at 47.0 cm. When families were mixed, the mean height was not significantly different from the mean height of the susceptible family. For all treatments, height increased with increasing distance and then slighly lowered (1.0 cm) at the farthest distance.

Georgia

There were no significant single family differences at any distance (Table 7) even though there were large differences in family means (Table 8). The large block to block variation for each family inflated the error term and a non-significant F-value was obtained. Single family means varied from 22.1 cm to 35.9 cm. SC-26 was the shortest while MS-5 was the tallest. The mean height for trees in the control plot was 36.6 cm. The mixtures of families did not significantly increase mean height above the susceptible family. For all treatments, height increased with inceasing distance and then decreased (2.8 cm) at the farthest distance.

There was no location or location x treatment interaction (Table 9). The mean height of the Mississippi planting was 38.4 cm compared to 32.6 cm for the Georgia planting. Family MS-5 was the tallest family at both locations. This family had the second shortest height after five years in Louisiana (Snyder 1977, Derr 1971).

Weather

Average temperatures were similar for both the Mississippi and Georgia plantings (Fig. 3, 4). Temperatures were somewhat cooler during the second growing season at both locations. Relative humidities were higher in Georgia than in Mississippi. A large difference occurred between locations for the amount of rainfall each received. During the 18 month duration of the test, the Mississippi planting received 169 cm

Table 7. Analysis of variance tables with mean squares for disease variables and height at four distances for six single family treatments of longleaf pine exposed to Scirrhia acicola for two growing seasons in Georgia.

Source	d.f.	YMAX [a] Distance (m)				TBEG [c] Distance (m)				TMAX [d] Distance (m)			
		0.3	1.2	2.1	3.0	0.3	1.2	2.1	3.0	0.3	1.2	2.1	3.0
Blocks	3	2.2	13.0	23.7* [b]	42.4*	1.7	8.4	5.4	6.5	8.0	5.2*	2.1*	0.2
Treatments	5	0.7	13.2	12.2	31.2*	1.5	12.9*	2.6	15.0	17.7*	1.6	0.4	0.5
Error 1	15	1.0	6.7	5.4	9.2	1.1	3.7	4.2	8.1	3.2	0.8	0.3	0.8
Transects	3	0.5	2.8	5.8	8.1	1.8	8.6	5.2	12.4*	0.3	1.2	0.2	0.7
Treatment x transects	15	0.8	2.4	3.1	4.7	1.2	3.3	4.8	1.6	2.8	2.1	0.3	0.5
Error 2	54	0.9	3.7	6.9	8.2	1.0	5.4	4.8	4.0	2.8	1.0	0.4	0.5

Source	d.f.	YRATE [e] Distance (m)				LESION NO. [f] Distance (m)				HEIGHT [g] Distance (m)			
		0.3	1.2	2.1	3.0	0.3	1.2	2.1	3.0	0.3	1.2	2.1	3.0
Blocks	3	.07	0.7	1.5	0.3	21330	579.5	161.7	113.4	640.2	1249	757.7	855.2
Treatments	5	.42	0.3	0.5	1.8	8004	584.3	75.3	121.3	640.2	958	652.5	127.2
Error 1	15	.31	0.9	1.4	5.2	21755	660.9	166.7	118.4	370.6	616	554.5	364.2
Transects	3	.17	1.1	0.3	5.1	15559	783.3	221.6	67.3	426.6	1275*	360.6	70.1
Treatment x transect	15	.34	0.6	1.8	3.0	8066	587.8	114.8	103.3	230.6	527	220.0	387.4
Error 2	54	.21	1.1	1.5	2.8	9768	461.4	144.1	104.9	239.4	352	319.2	290.7

Table 7--extended.

[a] Maximum percent needle dieback.

[b] Asterisks indicate the probability (P=0.05) of a value larger than F.

[c] Time (wk) when needle dieback was first observed.

[d] Time (wk) when YMAX was observed.

[e] Rate of percent needle dieback increase per week.

[f] Lesion numbers observed 18 wk after epidemic initiation.

[g] Second-year height (cm).

Table 8. Means by distance from initial infection focus of disease variables and height for single and multiple family treatments of longleaf pine exposed to Scirrhia acicola for two growing seasons in Georgia.

Treatment	Distance (m)	YMAX[ry] (%)	TBEG[sy] (wk)	TMAX[ty] (wk)	YRATE[u]	LESION[v] NO.	HEIGHT[w] (cm)	b_1[x]
MS-6 (R)[q]	0.3	98	19.8	71.4a	.019	125.9	19.5	.699
MS-4 (I)		99	17.4	67.8ab	.021	148.4	28.6	1.000
MS-5 (S)		98	16.8	66.6bc	.021	186.3	26.3	1.160
MS-14 (R)		97	17.4	63.0c	.025	149.0	28.9	1.040
SC-6 (I)		98	16.8	63.0c	.022	140.1	29.2	1.020
SC-26 (S)		94	14.4	52.8d	.026	174.4	13.8	1.320
MS-6 + MS-5		100	16.8	58.8	.026	182.5	27.6	1.190
MS-14 + MS-5		97	16.8	62.4	.022	219.6	18.8	1.050
MS-6 + SC-26		98	19.8	64.8	.023	118.3	25.4	.936
MS-14 + SC-26		98	15.0	61.2	.023	179.4	14.5	1.210
Control		83	52.8	72.6	.049	13.1	36.5	.167
$\bar{x}$[z]		98	16.8	63.0	.022	162.4	23.3	1.060
MS-6 (R)[q]	1.2	76	45.6a	74.4	.032	13.2	37.0	.156
MS-4 (I)		75	36.6ab	74.4	.025	11.6	36.7	.221
MS-5 (S)		96	32.4b	70.2	.028	26.0	42.6	.283
MS-14 (R)		83	39.6ab	75.6	.029	12.7	38.2	.143
SC-6 (I)		92	34.2b	73.2	.029	20.9	40.8	.287
SC-26 (S)		91	31.2b	71.4	.026	22.3	20.9	.343
MS-6 + MS-5		90	32.4	70.8	.025	13.2	43.1	.214
MS-14 + MS-5		86	34.8	72.6	.026	13.1	29.7	.389
MS-6 + SC-26		90	33.6	72.6	.028	26.5	41.7	.328
MS-14 + SC-26		95	26.4	70.8	.022	69.6	28.9	.409

Table 8--continued.

Treatment	Distance (m)	YMAX[ry] (%)	TBEG[sy] (wk)	TMAX[ty] (wk)	YRATE[u]	LESION[v] NO.	HEIGHT[w] (cm)	b_1[x]
Control	1.2	88	52.8	70.8	.051	5.1	43.9	.087
$\bar{x}$[z]		87	34.8	72.0	.028	22.9	36.0	.278
MS-6 (R)[q]	2.1	67	48.6	73.8	.028	11.5	32.0	.240
MS-4 (I)		76	46.8	75.6	.036	7.9	40.0	.234
MS-5 (S)		83	46.8	73.8	.036	8.5	39.6	.144
MS-14 (R)		81	46.8	74.4	.035	6.2	40.0	.093
SC-6 (I)		76	49.8	74.4	.036	5.2	36.0	.109
SC-26 (S)		94	42.6	73.2	.035	7.4	23.9	.201
MS-6 + MS-5		76	40.8	75.6	.024	12.3	41.1	.158
MS-14 + MS-5		76	43.2	75.6	.030	8.8	30.9	.176
MS-6 + SC-26		88	48.0	73.2	.042	16.5	43.1	.187
MS-14 + SC-26		91	38.4	72.6	.032	20.4	29.1	.310
Control		78	55.8	74.4	.056	2.6	41.2	.057
$\bar{x}$[z]		81	45.0	74.4	.034	10.4	35.6	.185
MS-6 (R)[q]	3.0	57b	58.2	76.2	.033	2.9	33.2	.044
MS-4 (I)		63b	47.4	73.8	.032	3.2	27.3	.479
MS-5 (S)		76ab	41.4	74.4	.032	4.6	35.0	.141
MS-14 (R)		64b	51.6	72.6	.031	3.9	33.2	.150
SC-6 (I)		67b	50.4	75.6	.036	3.2	32.5	.076
SC-26 (S)		96a	44.4	74.4	.045	10.1	29.7	.495
MS-6 + MS-5		84	51.0	73.2	.052	6.6	49.8	.143
MS-14 + MS-5		53	52.8	73.2	.024	2.3	28.8	.132
MS-6 + SC-26		89	49.2	77.4	.058	3.6	46.4	.033

Table 8--continued.

Treatment	Distance (m)	YMAX[ry] (%)	TBEG[sy] (wk)	TMAX[ty] (wk)	YRATE[u]	LESION[v] NO.	HEIGHT[w] (cm)	b_1[x]
MS-14 + SC-26	3.0	75	45.6	72.6	.033	4.3	24.8	.144
Control		77	45.0	75.6	.034	5.9	24.7	.212
$\bar{x}$[z]		72	49.2	74.4	.035	4.5	34.1	.184

[q] R, I, and S = resistant, intermediate, and susceptible to brown spot needle blight, respectively.

[r] Maximum percent needle dieback.

[s] Time (wk) when needle dieback was first observed.

[t] Time (wk) when YMAX was observed.

[u] Rate of percent needle dieback increase per week.

[v] Lesion numbers observed 18 wk after epidemic initiation.

[w] Second-year height (cm).

[x] Linear coefficient of a fitted polynomial based on the first growing season percent needle dieback.

[y] Numbers followed by the same letter are not significant (P=0.05) according to Duncan's multiple range test. Multiple family treatments were not included in this test.

[z] Mean of single family treatments only.

Table 9. Analysis of variance table with mean squares for disease variables and height over locations for six single family treatments of longleaf pine exposed to Scirrhia acicola for two growing seasons.

Source	d.f.	YMAX [a]	TBEG [b]	TMAX [c]	YRATE [d]	LESION NO. [e]	HEIGHT [f]
Locations	1	319.2	357.5*	23.7	1.6	191679*	7360
Blocks (locations)	6	54.5	14.1	6.9	1.6	4976	2187
Treatment x location	5	25.4* [g]	3.6	3.7*	1.1	955	1973
Error 2	30	9.8	9.3	1.4	2.7	4044	1239

[a] Maximum percent needle dieback.

[b] Time (wk) when needle dieback was first observed.

[c] Time (wk) when YMAX was observed.

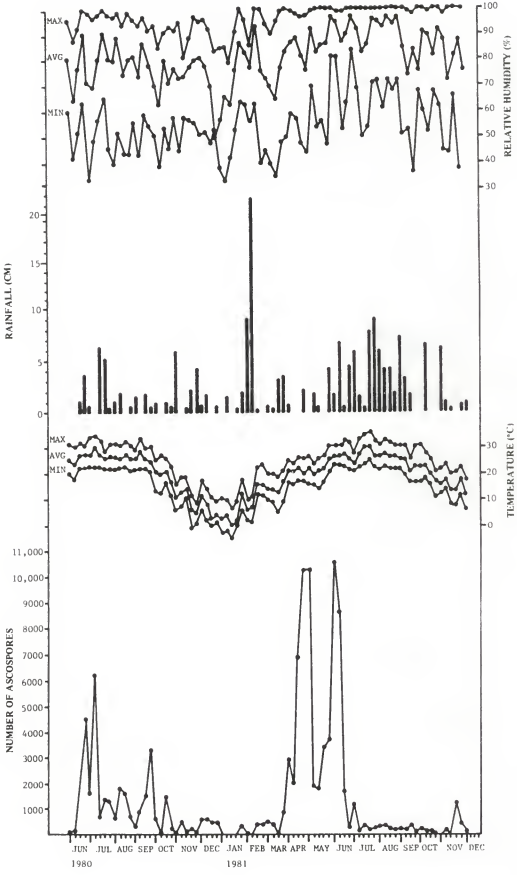
[d] Rate of percent needle dieback increase.

[e] Lesion numbers observed 18 wk after epidemic initiation.

[f] Second-year height (cm).

[g] Asterisks indicate the probability (P=0.05) of a value larger than F.

Fig. 3. Maximum, minimum, and average weekly percent relative humidity; total weekly rainfall; maximum, minimum, and average weekly temperature; and total number of Scirrhia acicola ascospores trapped per week; June 1, 1980 through December 31, 1981, on the Harrison Experimental Forest, near Saucier, Mississippi.


MAX
AVG
MIN
RELATIVE HUMIDITY (%)
100
90
80
70
60
50
40
30
RAINFALL (CM)
20
15
10
5
0
MAX
AVG
MIN
TEMPERATURE (°C)
30
20
10
0
NUMBER OF ASCOSPORES
11,000
10,000
9000
8000
7000
6000
5000
4000
3000
2000
1000
JUN JUL AUG SEP OCT NOV DEC JAN FEB MAR APR MAY JUN JUL AUG SEP OCT NOV DEC
1980
1981

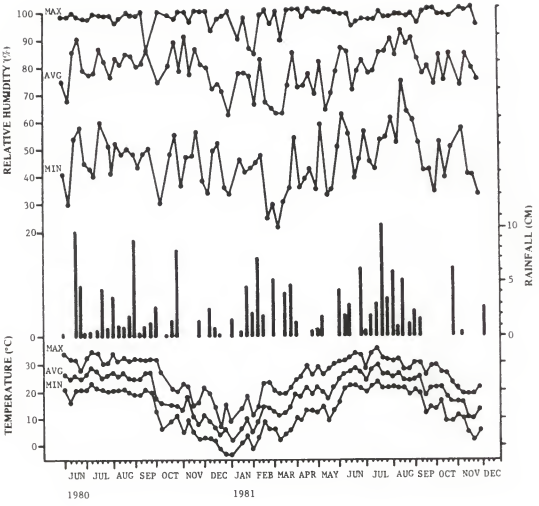


Fig. 4. Maximum, minimum, and average weekly percent relative humidity; total weekly rainfall; maximum, minimum, and average weekly temperature; June 1, 1980 through December 31, 1981, on the Southlands Experimental Forest, Bainbridge, Georgia.

of precipitation while the Georgia planting received 150 cm. However, the Georgia planting received over 13 cm more rainfall during the first growing season. As will be shown later, this more abundant rainfall significantly increased the amount of BSNB dieback at the Georgia planting by the end of the second growing season.

Spore Trapping

Ascospores

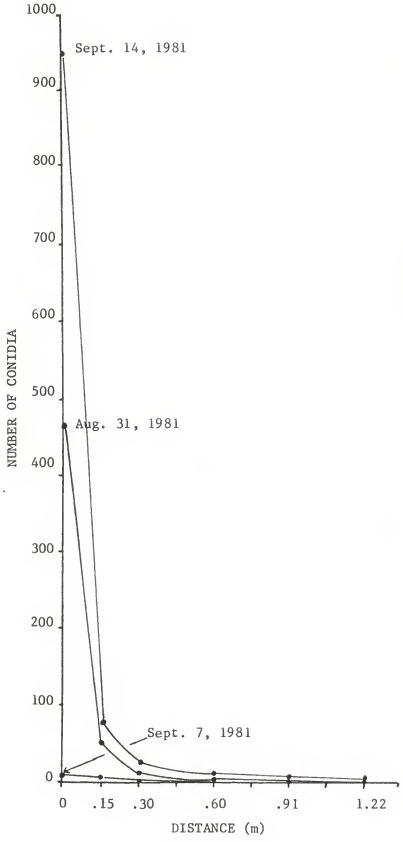
Ascospores were few in number the first growing season (May-December, 1980) (Fig. 3). The peak in late June and early July, 1980, was the ascospores coming from the severely-infected, necrotic needles placed in the center of the plots the last week in May, 1980 to initiate the epidemic (the planting sites were sanitized of S. acicola prior to planting and only disease-free seedlings were planted). Some more ascospores, though not as many as for July, 1980, were trapped in late September, 1980. These ascospores, too, came from the same necrotic needles because dieback had not been recorded by September, 1980 on the trees being measured.

However, by April-June, 1981 the number of ascospores trapped dramatically increased (Fig. 3). The ascospores were produced on the infected, dead needles of the sample trees. It took 4-5 months after the needle tissue died for ascospore production. Few ascospores were trapped in August, 1981 despite abundant rain.

Conidia

Conidia were trapped only when it rained. No conidia were trapped the week of September 21 when no rainfall was recorded. The dispersal gradients for conidia were steep (Fig. 5), indicative of local spread (Gregory et al. 1959).

Fig. 5. Dispersal gradients of Scirrhia acicola conidia trapped during three weeks in late summer, 1981, on the Harrison Experimental Forest, near Saucier, Mississippi.


1000
900
800
700
600
500
400
300
200
100
0
NUMBER OF CONIDIA
Sept. 14, 1981
Aug. 31, 1981
Sept. 7, 1981
0
.15
.30
.60
.91
1.22
DISTANCE (m)

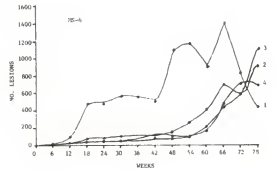
Disease Increase

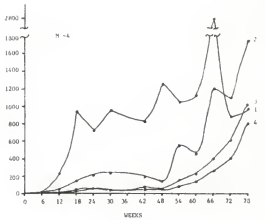
Lesion Numbers

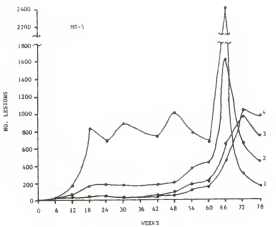
Mississippi. The number of lesions for all families first peaked at 18 weeks (October, 1980) after epidemic initiation (Fig. 6). Another peak occurred at 48 weeks (May, 1981). The highest number of lesions was observed at week 66 (September, 1981). Only trees at the closest distance had high numbers of lesions (>200) during the first growing season (through week 42) (Fig. 6). However, by the end of the second growing season, trees at all distances had high numbers of lesions. The decreases or negative rates in the progress curves were from needle dieback which resulted from coalescing lesions so that fewer lesions were observed. When new growth (needle flushes) began, lesion numbers increased until the needles became necrotic, again. Significant numbers (>25) of lesions did not occur in the control plots until late in the second growing season (September, 1981) when trees at all distances were affected simultaneously.

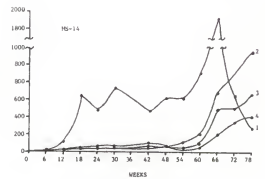
Needle dieback was first observed in the susceptible families 24 weeks after epidemic initiation. Since dieback is caused by the coalescing of lesions, lesion numbers were statistically analyzed at 18 weeks to minimize bias from dieback. Lesion numbers were inversely proportional to distance from initial infection focus. As a result, analyses were partitioned by distance. There were no significant treatment effects at any distance (Table 5), but there were significant transect effects. Brown spot needle blight lesions occurred nearly twice as frequent east of the initial infection focus in all plots at

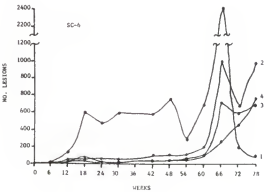
Fig. 6. Disease progress curves of lesion numbers at four distances from the initial infection focus in single and multiple family treatments of longleaf pine exposed to Scirrhia acicola for two growing seasons in Mississippi. 1 = 0.3 m, 2 = 1.2 m, 3 = 2.1 m, 4 = 3.0 m.

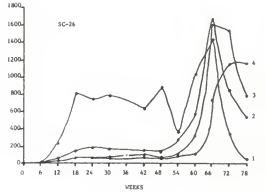

MS-6
NO. LESIONS
WEEKS

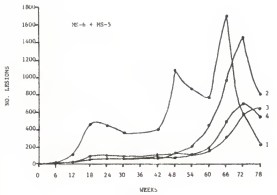

M -4
NO. LESIONS
WEEKS

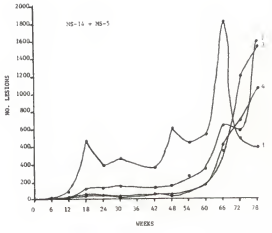

MS-5
NO. LESIONS
WEEKS

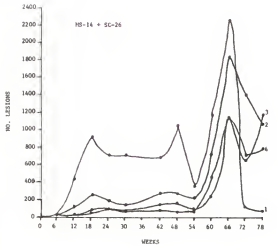

MS-14
NO. LESIONS
WEEKS

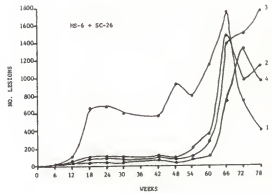

SC-4
NO. LESIONS
WEEKS


SC-26
NO. LESIONS
WEEKS









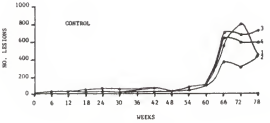


Fig. 6. continued.

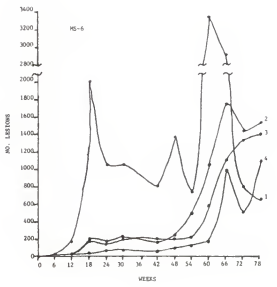
0.3 m and 1.2 m. The control had a mean of <2 lesions per tree. The mixtures of families did not significantly lower the mean number of lesions compared to the susceptible family mean (Fig. 6, Table 6), 18 weeks after epidemic initiation.

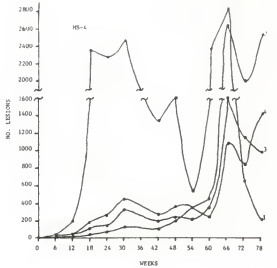
Georgia. Lesion numbers first peaked at 18 weeks, also. However, another peak of the same magnitude did not occur until weeks 60-66 (Fig. 7). There was a small peak 48 weeks after epidemic initiation. The closest distance had the highest numbers of lesions the first year, but by the second year trees at all distances were significantly affected by BSNB. The control did not have high numbers of lesions (>200) until the second growing season. Trees at all distances in the control plots were similarly affected (Fig. 7), perhaps indicative of an ascospore shower. The two treatments of mixed families involving MS-5 had the highest numbers of lesions. Family mixtures did not appreciably lower numbers of lesions except for the MS-6 + SC-26 treatment (Fig. 7).

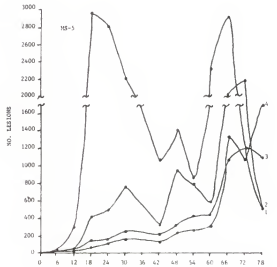
At 18 weeks there was a significant distance effect--lesion numbers were inversely proportional to distance from the initial infection focus. When data were analyzed by distance there were no significant treatment or transect effects (Table 7). Family mixtures did not lower the mean number of lesions (Table 8). The control had a mean of <7 lesions per tree.

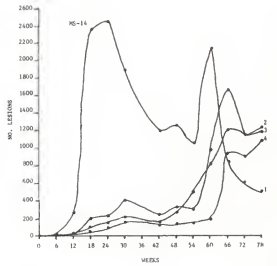
The average level of infection (lesion numbers) during the entire epidemic was about 1.5 times higher in Georgia than in Mississippi. Eighteen weeks after epidemic initiation, the Georgia planting had more than three times as many lesions as the Mississippi planting and this was statistically significant (Fig. 8A, Table 9).

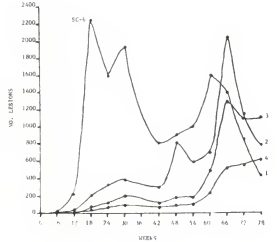
Fig. 7. Disease progress curves of lesion numbers at four distances from the initial infection focus in single and multiple family treatments of longleaf pine exposed to Scirrhia acicola for two growing seasons in Georgia. 1 = 0.3 m, 2 = 1.2 m, 3 = 2.1 m, 4 = 3.0 m.

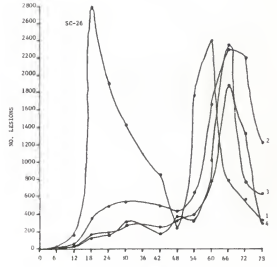

MS-6
NO. LESIONS
WEEKS

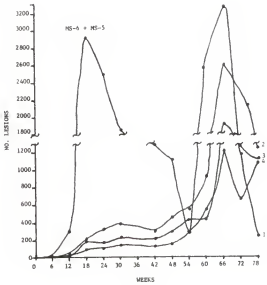

MS-4
NO. LESIONS
WEEKS

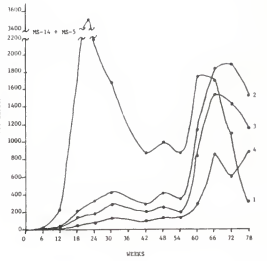

MS-5
NO. LESIONS
WEEKS

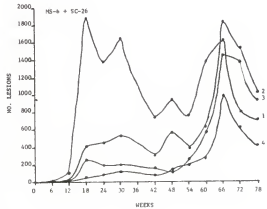

MS-14
NO. LESIONS
WEEKS

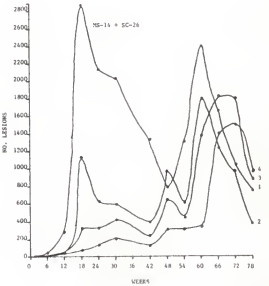

SC-15
NO. LESIONS
WEEKS


SC-24
NO. LESIONS
WEEKS









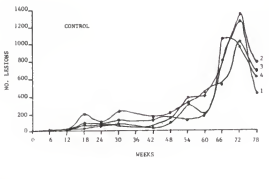
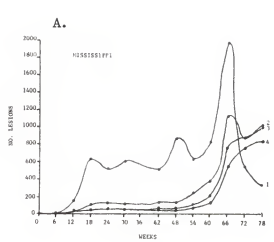
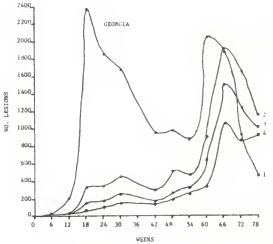
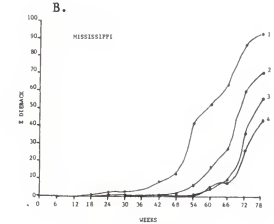


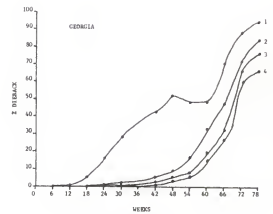
Fig. 7. continued.

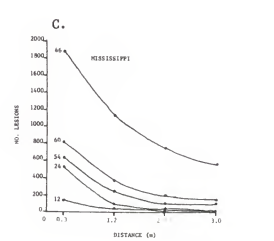
Fig. 8. Characterization of field epidemics of brown spot needle blight on six open-pollinated families of longleaf pine for two growing seasons in Mississippi and Georgia. A. Disease progress curves of lesion numbers at four distances from the initial infection focus. 1 = 0.3 m, 2 = 1.2 m, 3 = 2.1 m, 4 = 3.0 m. B. Disease progress curves of percent needle dieback at four distances from the intial infection focus. 1 = 0.3 m, 2 = 1.2 m, 3 = 2.1 m, 4 = 3.0 m. C. Disease gradient curves of lesion numbers at five different time periods. Numbers by curves indicate the weeks after epidemic initiation.

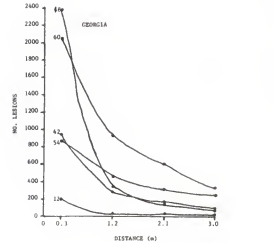

A.
MISSISSIPPI
NO. LESIONS
WEEKS


GEORGIA
NO. LESIONS
WEEKS


B.
MISSISSIPPI
% DISEASE
WEEKS


GEORGIA
% DISEASE
WEEKS


C.
MISSISSIPPI
NO. LESIONS
DISTANCE (m)


GEORGIA
NO. LESIONS
DISTANCE (m)

Two large peaks of lesion numbers occurred in the Georgia planting in October of each year. One large peak occurred in Mississippi during the second year (Fig. 8A).

Percent Needle Dieback

The disease progress curves of percent needle dieback were complex (Fig. 9, 10) with increases and decreases, particularly during the second growing season. The fluctuations in dieback were from the excessive removal of dead (BSNB-killed) needle fascicles and the rapid development of new needle growth or "flushes." The latter initially showed no BSNB dieback; hence the percent dieback actually decreased.

With the exception of the susceptible families, percent dieback never exceeded 10% during the first growing season (through week 42) in Mississippi (Fig. 9). In Georgia, percent dieback exceeded 10% in all families by week 24 (Fig. 10). At both locations dieback was inversely proportional to distance from initial infection focus. Percent dieback in Georgia reached higher levels earlier and maintained those levels by the end of the second growing season (Fig. 8B). No significant needle dieback was observed in the control plots at both locations until late summer of the second growing season, and curves were similar at all distances (Fig. 9, 10). This similarity in disease progress curves for all distances in the control plots was again interpreted as having arisen from an ascospore shower.

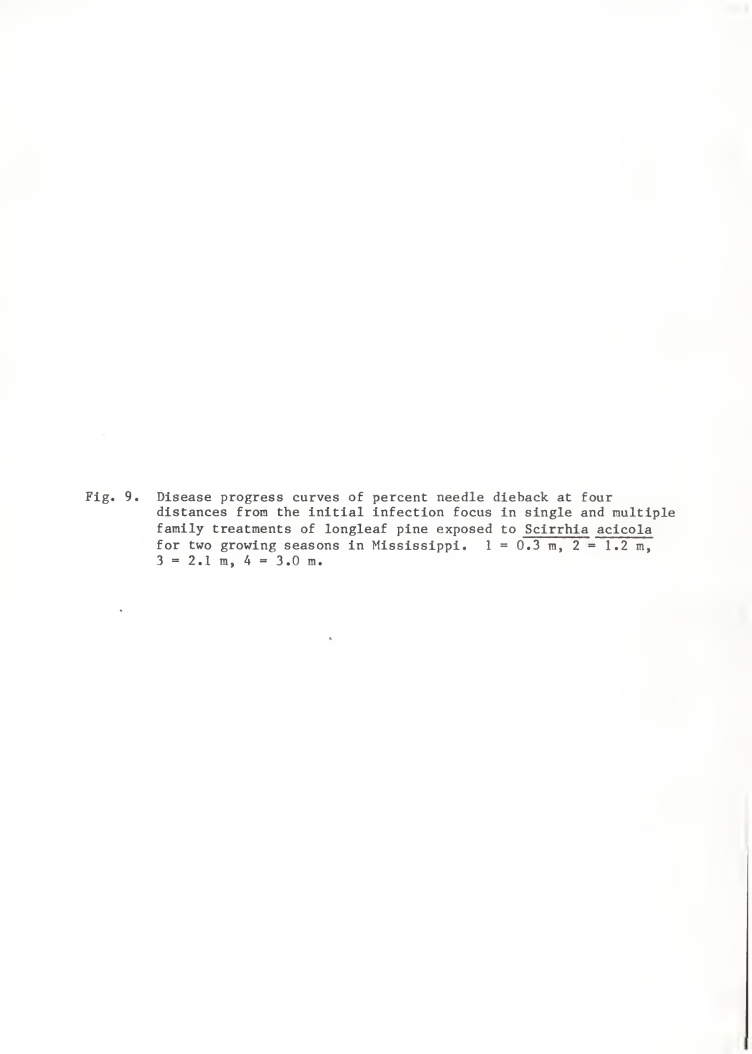
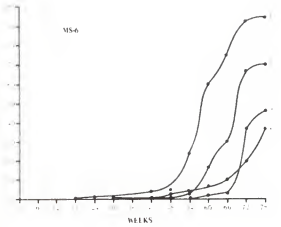
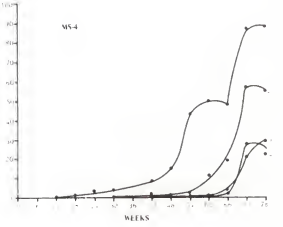
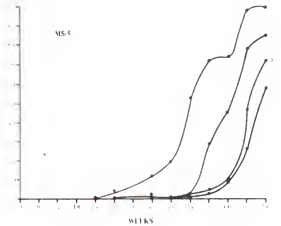
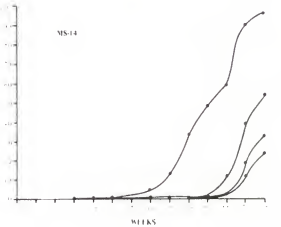


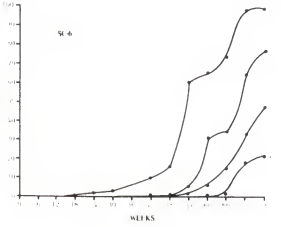
Fig. 9. Disease progress curves of percent needle dieback at four distances from the initial infection focus in single and multiple family treatments of longleaf pine exposed to Scirrhia acicola for two growing seasons in Mississippi. 1 = 0.3 m, 2 = 1.2 m, 3 = 2.1 m, 4 = 3.0 m.

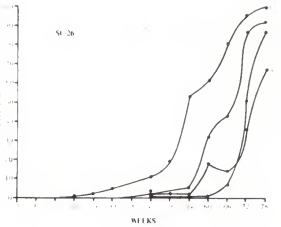

MS-6
DIEBACK
WEEKS

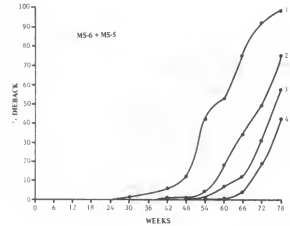

MS-4
DIEBACK
WEEKS

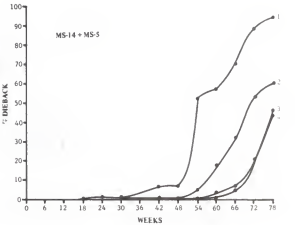

MS-5
DIEBACK
WEEKS

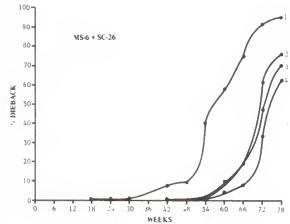

MS-14
DIEBACK
WEEKS

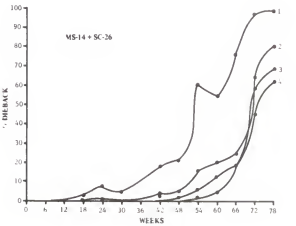

St-6
DIEBACK
WEEKS


St-26
DIEBACK
WEEKS









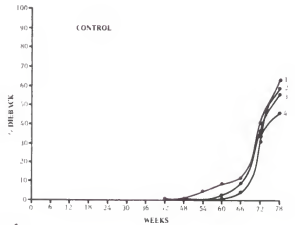
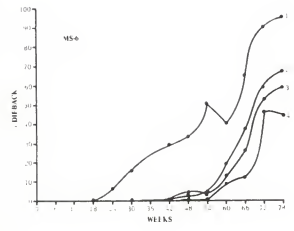
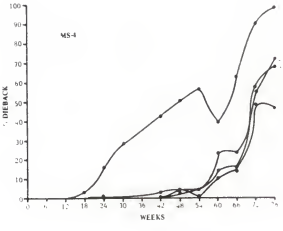
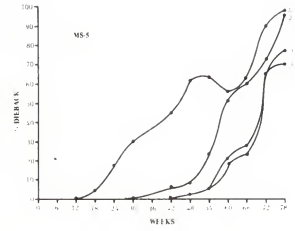


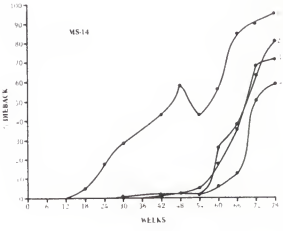
Fig. 9. continued.

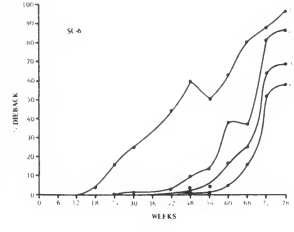
Fig. 10. Disease progress curves of percent needle dieback at four distances from the initial infection focus in single and multiple family treatments of longleaf pine exposed to Scirrhia acicola for two growing seasons in Georgia. 1 = 0.3 m, 2 = 1.2 m, 3 = 2.1 m, 4 = 3.0 m.

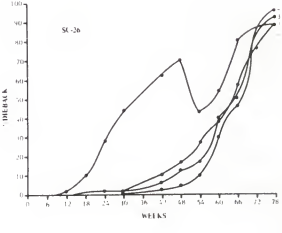

MS-6
% DIEBACK
WEEKS

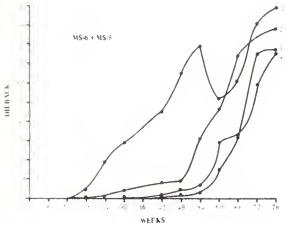

MS-4
% DIEBACK
WEEKS

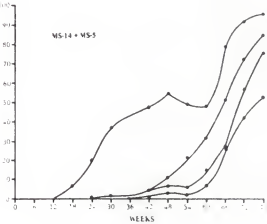

MS-5
% DIEBACK
WEEKS

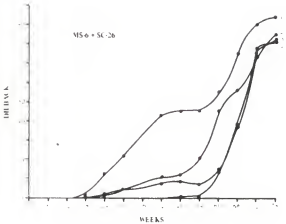

MS-14
% DIEBACK
WEEKS

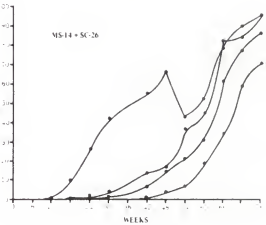

SL-6
% DIEBACK
WEEKS


SL-26
% DIEBACK
WEEKS









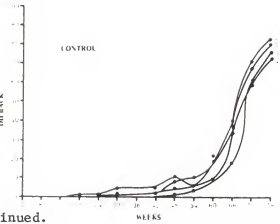


Fig. 10. continued.

Polynomial curve fitting

Since the disease progress curves of percent dieback were complex, only the curves for the first growing season (through week 48) were fitted with polynomials. Using polynomials of second degree or higher gave little or no increase in goodness-of-fit (R^2). Thus, a first degree polynomial (linear) with $b_o = 0$ was in close agreement with the actual first year curves. The typical polynomial form appeared as

$$y(t) = b_1 t$$

where y(t) was the predicted dieback at time t, and b_1 the linear polynomial coefficient. Analyses of the b_1 coefficients gave no significant family effects at both Mississippi and Georgia. Minimum needle dieback during the first growing season precluded differentiating between families (Table 6, 8). At both locations dieback developed more rapidly closest to the initial infection focus than at the other distances (Fig. 8B). This was more evident in Georgia. Location differences were significant--the mean b_1 at Georgia was four times greater than that at Mississippi.

Curve elements

Maximum percent dieback (YMAX). *Mississippi*. Family effects were significant for YMAX at all distances except at the closest distance (Table 5). Apparently inoculum levels were so high at 0.3 m that the resistance of all treatments was overcome. The two susceptible single family treatments, MS-5 and SC-26, had the highest YMAX at all distances where treatment effects were significant (Table 6). The resistant family, MS-14, was the most resistant at the outer three distances. There was a treatment x transect interaction at the 1.2 and 2.1 m distances from the inconsistent behavior of treatment SC-6. This

family had a large amount of dieback north of the initial focus at 1.2 m and 2.1 m, but significantly higher amounts of YMAX were observed east of the initial infection focus for the other families. Family means for YMAX over all distances ranged from 54-87%. Family mixtures lowered the overall mean of YMAX compared to the susceptible family, but not significantly so. YMAX decreased as distance increased from the initial infection focus (Table 6).

Georgia. Family effects for YMAX were significant only at 3.0 m (Table 7). Families MS-5 and SC-26 again had the highest YMAX, while resistant MS-6 had the lowest YMAX values (Table 8). Over all distances, treatment means ranged from 74-93%. The YMAX decreased as distance increased from the initial infection focus for all families (Table 8). The west side of the planting (block 1) had significantly less dieback. Family mixtures in Georgia also did not result in any lowering of YMAX compared to the susceptible families.

There was a significant location x family effect although the location effect itself was not significant (Table 9). The Georgia planting had a mean YMAX of 83% compared to a mean of 70% for the Mississippi planting. Family MS-6 had the third highest YMAX in Mississippi, but had the lowest YMAX in Georgia.

Time of beginning needle dieback (TBEG). *Mississippi*. Dieback began significantly earlier east of the initial infection focus at all distances except at 1.2 m (Table 5). The trees in the east transect also had more lesions. Single family treatment effects were significant only at 3.0 m (Table 5). There was also a significant transect x family interaction at 3.0 m because the susceptible SC-26 exhibited dieback in

the west transect much later than would be expected. The reason for this is unknown. The susceptible MS-5 and SC-26 families had the earliest times, while resistant MS-6 and MS-14 had the latest times (Table 6). Over all distances, family means for TBEG ranged from 40.8-48 weeks. Family mixtures did not significantly delay the time at which dieback began. The trees in the control plots did not have dieback until 67 weeks. This value was 35 weeks later than the resistant families. The values for TBEG increased as distance increased from the initial infection focus.

Georgia. Treatments were only significant at 1.2 m (Table 7). Families MS-5 and SC-26 again had the earliest times and MS-6 and MS-14 the latest (Table 8). Single family means over all distances ranged from 33-43.2 weeks. Needle dieback began significantly earlier northwest and southwest of the initial focus at 3.0 m. The trees in the control plots did not exhibit needle dieback until 53 weeks--33 weeks later than the resistant families (Table 8). Again, family mixtures did not significantly delay the time at which dieback was observed. Values of TBEG increased as distance increased from the initial infection focus (Table 8).

There was a significant location effect (Table 9). The Georgia planting had dieback 8.4 weeks earlier than the Mississippi planting.

Time of maximum needle dieback (TMAX). *Mississippi*. There were no significant single family treatment differences for TMAX. Transects were significant at 0.3 m (Table 5). The north and east transects reached maximum dieback earlier as did the east side of the planting

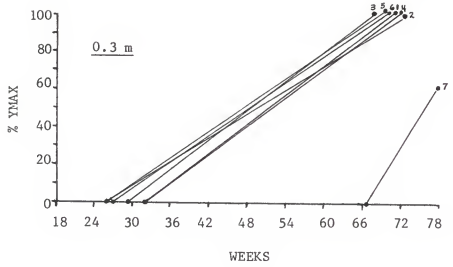
(block 4). As expected the trees in the control plots reached maximum dieback later (Table 6). Family mixtures had no effect on TMAX. Values of TMAX increased as distance increased from initial infection focus (Table 6).

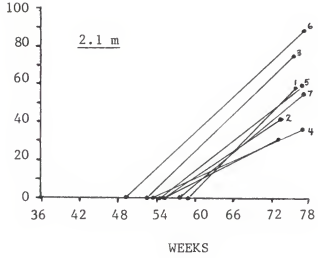
Georgia. Significant single family effects occurred at 0.3 m (Table 7). Family SC-26 reached its maximum dieback later than all other families. Maximum dieback was attained later on the west side of the planting (block 1). Family mixtures had no effect on TMAX (Table 8). The tree in the control plots did not have a later TMAX than the resistant MS-6. Values of TMAX increased as distance from initial infection focus increased (Table 8).

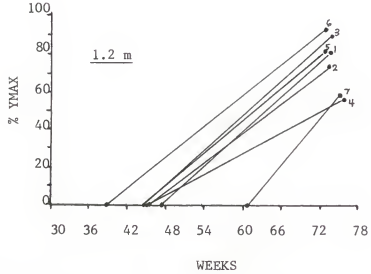
There was no location effect but there was a location x family effect (Table 9). In terms of rank, SC-26 had a later TMAX at Mississippi but the earliest TMAX at Georgia. The reverse was true for MS-4.

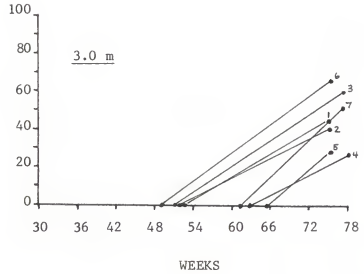
Rate of percent needle dieback increase (YRATE). *Mississippi*. There were no significant family differences at any distance for YRATE (Table 5). Separation of families was best at the farthest distance, 3.0 m (Fig. 11). Generally, families which had an earlier TBEG had a higher YMAX, while those with a later TBEG had a lower YMAX. Families MS-5 and SC-26 had the fastest YRATE values while MS-6 and MS-14 had the lowest. Family means over all distances ranged from .026-.033. Family mixtures of resistant and susceptible families did not significantly lower the YRATE (Table 6). The control had the fastest YRATE over all distances.

Fig. 11. Rate of percent needle dieback increase (YRATE) by distance from initial infection focus in single family treatments of longleaf pine exposed to Scirrhia acicola for two growing seasons in Mississippi. 1 = MS-6, 2 = MS-4, 3 = MS-5, 4 = MS-14, 5 = SC-6, 6 = SC-26, 7 = Control.


0.3 m
% YMAX
WEEKS
100
80
60
40
20
0
18 24 30 36 42 48 54 60 66 72 78


2.1 m
WEEKS
100
80
60
40
20
0
36 42 48 54 60 66 72 78


1.2 m
% YMAX
WEEKS
100
80
60
40
20
0
30 36 42 48 54 60 66 72 78


3.0 m
WEEKS
100
80
60
40
20
0
30 36 42 48 54 60 66 72 78

Georgia. Family effects were not significant, but family differences were most evident at the farthest distance (Fig. 12). The susceptible families, MS-5 and SC-26, had the fastest YRATE values and largest YMAX values at this distance, while the resistant families, MS-6 and MS-14, had the lowest YMAX values. Over all distances family means ranged from .028-.033. Family mixtures had variable effects in Georgia. When MS-6 was included the rates increased, and when MS-14 was included rates decreased. The control treatment had the fastest YRATE for all distances except at 3.0 m.

There were no significant location or location x treatment effects. At both locations YRATE tended to increase with increasing distance from the initial infection focus.

Disease Spread

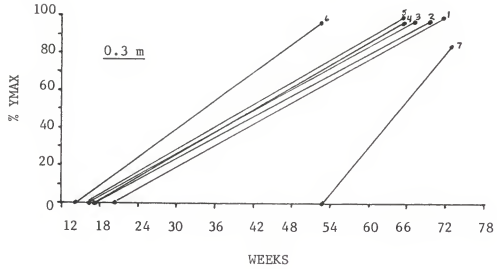
Disease Gradients

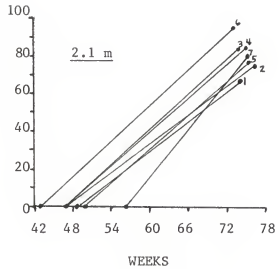
Lesion numbers

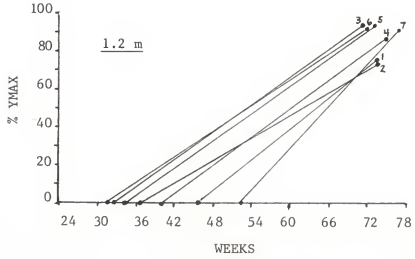
Lesion numbers ($\log_{10}$) at 18 weeks after epidemic initiation were used to calculate slopes of gradient curves by family since needle dieback commenced at 24 weeks in susceptible families. There were o significant family effects at either Mississippi or Georgia. However, there was a significant location effect--the gradient was significantly steeper in Georgia than in Mississippi: -1.60 vs -1.28 (Table 10). As expected, the control plots had no gradient consistent with the other families since the former was not inoculated.

Gradient curves for untransformed lesion numbers by location were steeper (more negative slopes) for Georgia than for Mississippi (Fig. 8C, Table 11). This was especially true after 12 weeks.

Fig. 12. Rate of percent needle dieback increase (YRATE) by distance from initial infection focus in single family treatments of longleaf pine exposed to Scirrhia acicola for two growing seasons in Georgia. 1 = MS-6, 2 = MS-4, 3 = MS-5, 4 = MS-14, 5 = SC-6, 6 = SC-26, 7 = Control.


0.3 m
% YMAX
WEEKS


2.1 m
WEEKS


1.2 m
% YMAX
WEEKS

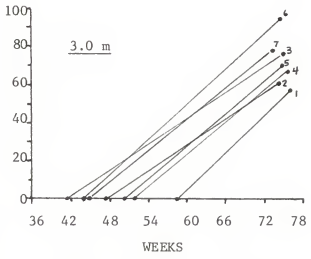

3.0 m
WEEKS

Table 10. Mean slope coeffecients (b) of gradients for lesion numbers ($\log_{10}$) 18 weeks after epidemic initiation with Scirrhia acicola for single and multiple family treatments of longleaf pine planted in Mississippi (HEF) and Georgia (SEF).

Treatment	Location	
	HEF	SEF
	--b--	
MS-6 (R)[z]	-1.21	-1.52
MS-4 (I)	-1.38	-1.67
MS-5 (S)	-1.20	-1.66
MS-14 (R)	-1.38	-1.64
SC-6 (I)	-1.22	-1.69
SC-26 (S)	-1.31	-1.44
MS-6 + MS-5	-1.13	-1.51
MS-14 + MS-5	-1.14	-1.94
MS-6 + SC-26	-1.32	-1.52
MS-14 + SC-26	-1.33	-1.46
Control	0.18	-0.08

[z] R, I, and S = resistant, intermediate, and susceptible to brown spot needle blight, respectively.

The curve flattened (becomes less negative) in Georgia from week 18 to week 54 (Table 11). This was caused by excessive needle dieback (resulting in fewer numbers of lesions) at the closest distance (Fig.8C). After week 66 in Mississippi and week 60 in Georgia, so much needle dieback occurred that secondary gradients dominated and the primary gradients became meaningless.

Percent needle dieback

Forty-eight weeks after epidemic initiation needle dieback was observed at the farthest distance from the center of all plots. Therefore, slopes of percent dieback gradients were calculated for each measurement period beginning at week 48 (start of second growing season) through the termination of the study at week 78 (Table 12, 13). The $\log_{10}$ x transformation was used instead of logit x because at 100% dieback levels the logit transformation gave erroneously steep slopes. The $\log_{10}$ transformed disease gradients did not depart from linearity as determined by regression analyses.

Mississippi. There were no significant family differences until week 78. The gradients became more steep (more negative) with time until at weeks 66-72 when the gradients began to significantly flatten (less negative slopes). The flattening of the gradient was due to the intensification of secondary foci of S. acicola during this time, notably at the second distance, 1.2 m (Fig. 9). By week 78 susceptible MS-5 and SC-26 had the flattest gradients, while the resistant families, MS-6 and MS-14, and intermediate MS-4 had the steepest gradients (Table 12). The secondary foci developed more rapidly in the susceptible families. The multiple family treatments with MS-5 had intermediate

Table 11. Mean slope coefficients (b) of gradients for lesion numbers (log_{10}) various weeks after epidemic initiation with Scirrhia acicola for longleaf pine planted in Mississippi and Georgia.

Mississippi		Georgia	
Weeks after epidemic initiation	Slope (b)	Weeks after epidemic initiation	Slope (b)
12	-0.781	12	-1.000
24	-1.020	18	-0.901
54	-0.908	42	-0.701
60	-0.755	54	-0.536
66	-0.508	60	-0.724

Table 12. Slope coefficients (b) for disease gradients of proportion needle dieback (log_{10}) from Scirrhia acicola for single and multiple family treatments of longleaf pine planted in Mississippi.

Treatment	Weeks after epidemic initiation					
	48	54	60	66	72	78[z]
			--b--			
MS-6 (R)[y]	-.031	-.157	-.322	-.361	-.298	-.234bc
MS-4 (I)	-.107	-.270	-.305	-.273	-.291	-.289ab
MS-5 (S)	-.138	-.314	-.377	-.344	-.280	-.135d
MS-14 (R)	-.086	-.222	-.304	-.345	-.368	-.302a
SC-6 (I)	-.122	-.350	-.364	-.366	-.223	-.293ab
SC-26 (S)	-.140	-.308	-.284	-.355	-.330	-.090d
MS-6 + MS-5	-.093	-.271	-.312	-.372	-.331	-.202
MS-14 + MS-5	-.057	-.323	-.331	-.355	-.312	-.193
MS-6 + SC-26	-.075	-.253	-.295	-.327	-.224	-.111
MS-14 + SC-26	-.143	-.310	-.258	-.273	-.193	-.128
Control	.003	.026	.036	.052	.014	-.067

[y] R, I, and S = resistant, intermediate, and susceptible to brown spot needle blight, respectively.

[z] Numbers followed by the same letter are not significant (P=0.05) according to Duncan's multiple range test. Multiple family treatments were not included in this test for week 78.

Table 13. Slope coefficients (b) for disease gradients of proportion needle dieback (log_{10}) from Scirrhia acicola for single and multiple family treatments of longleaf pine planted in Georgia.

Treatment	Weeks after epidemic initiation					
	48	54	60[z]	66[z]	72[z]	78[z]
			--b--			
MS-6 (R)[y]	-.215	-.301	-.187cd	-.260ab	-.177a	-.180a
MS-4 (I)	-.278	-.322	-.164d	-.258ab	-.165ab	-.168ab
MS-5 (S)	-.349	-.323	-.178cd	-.197bc	-.104bcd	-.095cde
MS-14 (R)	-.337	-.243	-.262ab	-.302a	-.130abc	-.116cd
SC-6 (I)	-.327	-.285	-.299a	-.302a	-.120abc	-.120bc
SC-26 (S)	-.306	-.164	-.100e	-.136d	-.001e	.022f
MS-6 + MS-5	-.353	-.394	-.181	-.144	-.083	-.080
MS-14 + MS-5	-.299	-.283	-.225	-.254	-.182	-.132
MS-6 + SC-26	-.268	-.258	-.235	-.188	-.047	-.042
MS-14 + SC-26	-.318	-.187	-.164	-.178	-.094	-.065
Control	-.023	.010	-.050	-.068	-.036	-.019

[y] R, I, and S = resistant, intermediate, and susceptible to brown spot needle blight, respectively.

[z] Numbers followed by the same letter are not significant (P=0.05) according to Duncan's multiple range test. Multiple family treatments were not included in these tests.

slopes while those involving SC-26 had slopes which were as flat or flatter than the susceptible families by themselves (Table 12). The control had flat gradients (very small slopes) at all times, since no point source of inoculum was located in these plots.

Georgia. There were significant family effects for weeks 60-78. The gradients began to significantly flatten (less negative) earlier at this location--during weeks 54-60. Families experienced severe defoliation (excessive removals) at 0.3 m while simultaneously producing new needle flushes (disease-free) that the gradients became less negative. Dieback actually declined (Fig. 10, Table 8). However, in 6-12 weeks time, after the new needles became infected and died from S. acicola, the gradients steepened (more negative) again (Fig. 10, Table 13). At that time, secondary foci developed and intensified such that by week 72 the gradients flattened once more (Fig. 10, Table 8, 13). Families MS-5 and SC-26 again had the flattest gradients by week 78. Secondary foci development increased so much that the gradient became positive for SC-26. The multiple family treatments had gradient slopes similar to the susceptible families. The control showed a weak gradient, since there was no point source of inoculum.

Gradients were significantly steeper (more negative) in Georgia than in Mississippi at week 48. Thereafter, Mississippi had steeper slopes due to the more severe secondary foci intensification in Georgia.

Rate of Spatial Disease Spread

Lesion numbers

The rate of spread in space (m/wk) was calculated starting at week 18 for both locations. There were no significant treatment or location effects. The rates ranged from 0.04-0.06 m/wk at both locations.

Percent needle dieback

The rate of spread for percent needle dieback was calculated for weeks 42, 48, and 54 at the Mississippi planting and for weeks 24, 30, and 42 for the Georgia planting. The trees in Georgia experienced needle dieback considerably earlier, hence, the time difference.

There were no treatment differences in the rate of spread for any measurement period at either location. Rates during the three time periods ranged from 0.10-0.14 m/wk in Mississippi, while at Georgia rates varied only from 0.06-0.11 m/wk. Thus, although intensities (YMAX) were different among families, the rates of spread outward were similar. Rates showed a slight tendency to increase with time. This could be due to interplot interference or spread from secondary foci, conditioned by rainfall.

Correlation of Tree Height and Disease

Correlations of height with the various disease variables at each location were not significant. The correlation coefficient of height vs YMAX was only -0.18 in Mississippi and only -0.05 in Georgia. Height correlated best with lesion numbers at 18 weeks: -0.46 in Mississippi and -0.45 in Georgia. Height was positively correlated with YRATE: 0.41 in Mississippi and 0.49 in Georgia. Correlations were positive since trees which did not experience needle dieback until the second growing season were taller and had larger YRATE values.

DISCUSSION

Brown spot needle blight severity was greater in Georgia than in Mississippi even though the quantity and quality of initial inoculum was similar at both locations. The difference may be the result of a more favorable environment for disease spread during the first year in Georgia. During the first growing season the Georgia planting experienced 13 cm more rainfall and for 11 weeks had maximum weekly percent relative humidity at 100%. At the Mississippi planting, however, the maximum weekly relative humidity reached 100% only for 2 weeks during this time period. Also during the two growing seasons, average and minimum weekly percent relative humidities were higher in Georgia than in Mississippi. Even though the Mississippi planting received over 30 cm more rainfall the second year, the Georgia planting maintained the higher disease levels because it was able to have more secondary cycles of BSNB infection during the first growing season.

Ascospores played a significant role during the latter half of the epidemic (weeks 48-78) at both locations, as evidenced by the abundant ascospores trapped at the beginning of the second growing season (Fig. 3), and the disease progress curves for the non-inoculated control treatments (Fig. 6-10). It is probable that Scirrhia acicola entered these plots as wind-disseminated ascospores. In addition, interplot interference was noted in some inoculated plots by the end of the second year--some trees at the edge of the plots (3.0 m) had more dieback than

trees on the same transect at 2.1 m (Fig. 3, 4). This was more evident in Georgia where disease was greater. Although the limited human traffic into the plots to measure could have caused the interference (by carrying conidia of S. acicola) this was not reflected in the disease progress curves for the control plots as very few lesions and virtually zero levels of needle dieback were recorded during the first year. If conidia were spread by humans, some needle dieback should have been observed the first year in the control plots. Likewise, the ascospores must have come from diseased needles within each planting rather than from 30+ year-old native longleaf pine across the road from each planting at each location. If the ascospores were from the mature longleaf pine stand, needle dieback should have been observed also during the first growing season in the control plots.

The fact that very few ascospores were trapped in August, 1981, is contradictory (Fig. 3) to previous reports (Kais 1971). However, August, 1981 was extremely hot (weekly maximum) in Mississippi (>30 C). This may have affected the dissemination of ascospores, for when the temperature (weekly maximum) dropped below 30 C, ascospores were trapped. Kais' (1971) study occurred during a relatively cool August: 24 C (weekly maximum). It appears that in late summer/early fall when temperatures are below 30 C (weekly maximum) and there is rain, fog, or dew, ascospores are disseminated. Siggers (1944) felt the quantity of ascospore inoculum was relatively limited. Nevertheless, their effectiveness is not diminished because they did have a profound impact on the pattern of later (second year) disease development at both locations in my plots.

Although progress curves for lesion numbers proved useful to explain biologically the course of the BSNB epidemic (Fig. 8A-C), they did not prove useful to assess BSNB resistance. The curves fluctuated greatly since the lesion numbers were biased by the amount of needle dieback. In addition lesion numbers alone did not express cumulative infection.

Progress curves of percent needle dieback were more suitable to assess resistance among the various longleaf pine families, since needle dieback is a more cumulative result of the disease. The curve elements identified from the progress of percent needle dieback curves were particularly useful.

Van der Plank (1968) stated that disease progress curves were the simplest and perhaps the best way to show how resistance in the host could be interpreted in terms of disease in the field. For example:

(i) If the infection rates ($\underline{r}$) do not vary between a resistant cultivar (R) and a susceptible cultivar (S), $\underline{r}_R = \underline{r}_S$, then for the resistant cultivar X_o will occur at a much later date (t_o) and ($X_o + X_{t+1}$) will be less.

(ii) If the infection rates do vary between a resistant and a susceptible cultivar, $\underline{r}_R \neq \underline{r}_S$, two ways to interpret the resistance are: (a) if $\underline{r}_R < \underline{r}_S$ then the times (t_o) at which X_o occurs for the two cultivars will be similar and $X_o + X_{t+1}$ will be less for the resistant cultivar, (b) if $\underline{r}_R > \underline{r}_S$ then X_o for the resistant cultivar will occur at a much later date (t_o) and $X_o + X_{t+1}$ will again be less for the resistant cultivar.

By using the BSNB curve elements identified in this study resistance to BSNB was expressed as (i); i.e., both resistant and susceptible longleaf families had similar YRATE values, but the resistant families had later TBEG values ($= t_o$) and lower YMAX values ($X_o + X_{t+1}$). Even though the calculation of YRATE implies that this parameter is linear over time, it should not be construed as such. The YRATE was the best that could be done with the way the data were collected for this study. It was very difficult or impossible to tally accurately BSNB infection on longleaf pine to conform to the definition of disease progress--cumulative infection--over the two years of this epidemic. Longleaf pine has an indeterminate growth pattern of innumerable fascicles coupled with numerous secondary cycles by the pathogen. Thus there were many fluctuations in the progress curves.

At both locations YRATE tended to increase with increasing distance from the initial infection focus. This may occur because of the method that YRATE was calculated:

$$\text{YRATE} = \frac{\text{YMAX}}{\text{TMAX} - \text{TBEG}}$$

Trees closest to the center of the inoculated plots (0.3 m) had low (early) TBEG values, but larger (lower) TMAX values due to the lack of rain and higher temperatures during the first growing season. This prohibited disease increase and spread. Because the denominator was large, lower YRATE values were obtained. On the other hand, trees farther from the inoculated plot centers and trees in the control plots had later TBEG values, i.e., the second growing season. During the second year background inoculum levels were higher than the first year, rainfall was more abundant, and temperatures were lower. With such

optimal conditions, the time between TBEG and TMAX was considerably shortened. As a result the denominator consisted of small values and this led to higher YRATE values for the trees farther from the initial infection focus and for the control.

For most of the curve elements large family variation from block to block often precluded the ability to distinguish statistically between single family treatments. Half-sib families probably introduced considerable variation in BSNB resistance and in height as indicated earlier by Derr (1971) and Snyder et al (1977). In spite of the large variation encountered the resistant families MS-6 and MS-14 consistently ranked as the best performers for each disease variable (curve element), while the susceptible families MS-5 and SC-26 always ranked the poorest. When significant single family differences did occur it was at distances farther from the center of the initial infection focus. I interpret this to mean that spore concentrations were greater near the center of the plot and prevented the separation of families. Another reason for the lack of family significance may be due to the inoculum. The inoculum may have been comprised of a population uniform in pathogenicity characteristics. If the inoculum was uniform and able to overcome most genes present in both resistant families, then one would not expect to see differences between resistant and susceptible families or any benefits form using mixed plots. Resistant families could be different in other components of resistance, but these may not be expressed if the interaction was with pathogen isolates able to overcome the genes responsible for the resistance.

The resistance displayed by the longleaf pine families primarily involved delaying the onset of needle dieback--tolerance. All families had similar lesion numbers 18 weeks after epidemic initiation; but some families (the susceptible ones) expressed needle dieback considerably earlier. Once dieback began, it proceeded at a fairly constant rate in all families. Perhaps each family has a tolerance threshold to a toxin produced by S. acicola. Once this threshold is reached, resistance is overcome and needle tissue begins to die. In histopathological examinations of S. acicola on longleaf pine needles, Jewell (in press) observed the presence of S. acicola hyphae in sympton areas as being very limited and the amount of host tissue damaged as being far out of proportion to the presence of the pathogen. Jewell interpreted this to mean that a toxin may be produced by S. acicola.

It should be said at this point that the resistance of longleaf pine to BSNB historically has been based on the visual estimation of needle dieback on individual trees. This, of course, is a measure of tolerance. Since the parents used in this study were selected on the basis of their mean progeny needle dieback percentages, it is reasonable to expect that the resistance I observed was tolerance to needle dieback. Thus, resistance may involve more than tolerance to dieback, but I could not detect it because of the criterion I used to select the parents for this study.

Shain and Franich (1981), working with P. radiata, found a toxin (dothistromin) produced by the closely related S. pini (Dothistroma needle blight fungus). The toxin caused the typical necrotic, red band symptoms on P. radiata. The needle tissue was killed in advance of hyphal penetration of P. radiata (Gadgil 1967).

Gregory's model for describing a disease gradient, $y = a/_{X}b$, when plotted as $\log_{10}$ y (disease severity) vs $\log_{10}$ X (distance from focal source) should yield a straight line with slope b. This pattern was observed repeatedly for the curves of lesion numbers and percent dieback. The transformed curves flattened (became less negative) over time at both locations, but the disease gradient patterns in Georgia reflected a more intense secondary foci development than in Mississippi--the gradients in Georgia became flatter earlier in the epidemic (Fig. 8C).

The outward spread of BSNB from the initial infection focus proceeded at similar rates among families. There were no significant family differences for the average rate of movement of lesion numbers or percent needle dieback although intensities were different. The resistance in longleaf pine to BSNB appears to affect intensity levels rather than rates of increase or spread.

A mixture of resistant and susceptible genotypes may achieve disease reductions in three ways:

(i) In a pure or single family plot having uniform susceptibility to a particular pathogen, the replacement of a proportion of these plants by resistant ones could reduce the amount of tissue which may become infected and this in turn would reduce the amounts of inoculum available for subsequent dispersal within the plot.

(ii) Replacement of susceptible plants by resistant ones could result in a decline in the density of the remaining susceptible plants and thus an increase in the average distance that inoculum has to travel between one susceptible plant and another; increased distance is often associated with factors that reduce the spread of inoculum.

(iii) Resistant plants may interfere with the passage of inoculum between susceptible plants.

The mixture of half-sib families (or varieties) in a non-random manner as was done in this study did not reduce disease levels from those encountered in susceptible single family plots. In fact, sometimes disease levels were higher in the multiple family plots. The failure of family mixtures to reduce disease incidence or spread can be attributed to the physical form of the longleaf grass stage seedling. Each seedling supports a very large number of fascicled needles (each

fascicle has three needles) which, in turn, provides an enormous surface area with the same genotype for S. acicola to infect and colonize. Thus, inoculum levels can increase rapidly (auto-infection) and a small epidemic can develop independently, unaffected by the presence or absence of a resistant neighbor tree. By comparison, in small grains an individual plant is small with a long and narrow shape, so that spores produced on it have a fair chance to land on a neighbor plant. This is one main reason why multilines and variety mixtures have worked so well in small grains. Small grains are inbred to produce specific cultivars susceptible only to a certain strain(s) of the pathogen such that spores produced on a cultivar by a strain of the pathogen are incapable of infecting a neighboring plant of a different cultivar. Thus, the spore cloud is diluted.

Splash dispersal of inoculum (as is primarily the case in BSNB) also encourages auto-infection (within-plant buildup) as it tends to limit dispersal to the immediate vicinity of the source plant. Allo-infection (between-plant transmission) is limited largely to those trees nearby (Robinson 1976). Thus, within the multiple family plots in this study the susceptible trees each ultimately acted as a disease spreader, overcoming the resistance of the resistant neighbor tree. Another disadvantage was the half-sib nature of the longleaf seedlings. This in itself created tree-to-tree variability within each family: a resistant parent can have resistant progeny, but more importantly, susceptible progeny as well. Resistant and susceptible half-sib family mixtures in longleaf pine did not appear to be a useful strategy to slow the BSNB epidemic in time or space.

Height was not significantly correlated with the various BSNB disease variables by the end of the second growing season. There were probably three contributing factors for this. First, to assure 100% planting survival in the field plots, vigorous nursery-grown seedlings were planted. Seedlings which are small, or undersized, are prone to planting death. Second, weed control within the plots in the form of hoeing, allowed virtually all seedlings to come out of the grass stage by the end of the second growing season. Longleaf seedlings are very intolerant of competing vegetation and will remain in the grass stage if the vegetation is not removed. Even though weed control allowed conidia to be rain-splashed at greater distances (thus assuring maximum spread of the fungus) the longleaf seedlings had the greater advantage. Third, the lack of sufficient rainfall during the first growing season did not allow for adequate BSNB increase to affect first-year seedlings. Third-year measurements of height growth and BSNB severity may allow for significant correlations between height and BSNB severity.

CONCLUSIONS

1. Resistance to BSNB in the half-sib longleaf pine families grown in Mississippi and Georgia was primarily a result of delaying the onset of needle dieback caused by S. acicola. Some families were very tolerant of lesion numbers, while others were not and expressed needle dieback quite early in the epidemic.

2. Resistant, intermediate, and susceptible longleaf pine families had similar rates of disease spread outward from initial infection foci. Initial and final disease intensities were different, but rates were not affected by resistance.

3. Progress curves of lesion numbers showed many fluctuations due to needle dieback.

4. Progress curves of percent needle dieback had some fluctuations. This was caused by excessive removal of BSNB-killed needles, coupled with rapid, new needle growth (flushes).

5. Curve elements of the percent dieback progress curves were helpful to compare families. The element TBEG (time at which needle dieback was first expressed) was always later for resistant families compared to susceptible families by as much as 5-7 weeks.

6. Disease gradient curves for numbers of lesions and percent needle dieback were steep (negative slopes), indicative of a rain splash-disseminated (local) disease. Gradients flattened (became less negative) with time due to secondary foci development within the plots.

7. Interplot interference caused by S. acicola ascospores (wind-disseminated) may have contributed to this intensification of secondary foci.

8. Ascospores were responsible for the BSNB observed in the control plots at both locations.

9. Resistant and susceptible family mixtures did not reduce disease levels or rates at either location.

10. Severity of brown spot needle blight reached higher levels in Georgia than in Mississippi. The environment was more favorable for disease development in Georgia.

11. Very little family x location interaction occurred. Only the variables YMAX and TMAX showed any statistical significance for this interaction.

Future epidemiological work with BSNB on longleaf pine should employ full-sib families to control the family x block variation. Ideally, clonal material should be used in spread plots. However, vegetatively propagating longleaf pine seedlings en masse is very difficult. In addition, percent needle dieback of entire plants should be recorded rather than just lesion numbers or percent dieback on a sample of fascicled needles. Lesion counts are time-consuming and labor-intensive, while estimating the amount of needle dieback on a whole plant is fast and more accurate to measure disease impact. Currently, this method is in use. The rate of lesion expansion, particularly as it relates to tolerance to needle dieback, should be investigated.

Future research to control BSNB should also include examination of the relationship between rapid, early growth in height; genetic resistance; and silvicultural techniques (vegetation control). This is a complex relationship but could be evaluated by using fungicide-sprayed vs non-sprayed plots and rough vs burned or cleared planting sites. It is often asked, "Is genetic resistance necessary if one can achieve rapid early growth in height in the pine by the use of proper silvicultural techniques?" This is an important question to answer in future research.

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I certify that I have read this study and that in my opinion it conforms to acceptable standards of scholarly presentation and is fully adequate, in scope and quality, as a dissertation for the degree of Doctor of Philosophy.

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